South African Medical Journal

S.-A. Tydskrif vir Geneeskunde

Organ of the Medical Association of South Africa

Vakblad van die Mediese Vereniging van Suid-Afrika

Incorporating the South African Medical Record and the Medical Journal of South Africa

Waarby ingelyf is die South African Medical Record and the Medical Journal of South Africa.

Registered at the General Post Office as a newspaper

By die poswese as nuusblad geregistreer

Cape Town, 2 January 1954
Weekly 2/6

Vol. 28 No. 1

Kaapstad, 2 Januarie 1954
Weekliks 2/6

IN THIS ISSUE — IN HIERDIE UITGAWE

South African Medical Journal

Suid.Afrikaanse Tydskrif vir Geneeskunde

P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 2 January 1954 Weekly 2/6 | Vol. 28 No. 1 | Kaapstad, 2 Januarie 1954 Weekliks 2/6

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Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

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Weekly 2/6 Weekliks 2/6

DISTURBANCES OF MITOTIC PROCESSES AND TERATISM

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In recent years clinical and experimental evidence in connection with congenital malformations and foetal deaths has thrown much light on the causes of teratism. The information collected suggests to us clear indications as to the causes and prevention of a large percentage of malformations and foetal deaths.

In the course of the last 5 or 6 decades experimental embryologists have produced various types of malformations and monsters in plants and aquatic animals by disturbing mitosis in embryos by means of chemical substances. These experiments, and especially Gregg's observations [1] in connection with congenital abnormalities in the offspring of pregnant mothers who suffered from rubella, turned our thoughts to the possibility of teratism in man being caused by the action of certain drugs and poisons shortly before or during pregnancy. It has become increasingly clear [3, 8, 15] that heredity is not the only factor which plays a rôle in malformations. Talbot [3] and Grebe and Windorfer [15] believe that most congenital abnormalities are not of an hereditary nature but are acquired *in utero*. According to Richet and Rymer [2] congenital abnormalities occur in 1–2% of normal individuals and some 300 different types are known. These include not only abnormalities of the organs but also congenital troubles associated with (1) the endocrine system (myxoedema, congenital diabetes, thymic hypertrophy, a tendency to obesity) and (2) diencephalo-hypophyseal troubles (dwarfishness, infantilism, etc.). It is obvious that the type and degree of damage done to the embryo depend on the force of the insult, the stage of gestation at which it takes place, and the period during which it acts. However, recent studies on cortisone by Fraser and his associates have shown that this hormone is capable of destroying foetal tissues which are already fully formed (cleft palate).[4]

It is proposed to deal here with congenital deformities and foetal death in so far as they are caused by (a) vitamin deficiencies and (b) toxic agents, including diseases and conditions which induce anoxia.

(A) VITAMIN DEFICIENCIES

Vitamin A. Hale [6] observed that sows on a diet deficient in vitamin A gave birth to young showing microphthalmia or anophthalmia, and other malformations, including harelip, cleft palate, and accessory ears. These observations were made on sows fed on a vitamin A deficient diet for 150 to 200 days before mating and during the first 30 days of gestation. Supplementation of the diet with vitamin A and D after 30 days gestation failed to correct the above malformations. However, when these vitamin supplements were given to these sows throughout the whole period of gestation the offspring were normal. Zilva *et al.*[7] made similar observations on sows on vitamin A deficient diets many years before. Investigations conducted by Warkany and Nelson [51] confirmed these observations of Zilva *et al.* and of Hale. According to Wilson *et al.*[10] vitamin A deficiency in the pregnant rat causes death of the foetus and various malformations in the eyes, the heart-blood-vessel system and the genito-urinary system. They were able to prevent foetal death and the abnormalities by the addition of 150 μg. carotin/week to the diet before and during pregnancy.

Not only hypovitaminosis A but also hypervitaminosis A may produce foetal abnormalities. Rodahl (*quoted by* Cohlan [48]) and Moore and Wang [50] reported that excess of vitamin A in the diet of pregnant rats caused a decrease in the number of litters and foetal resorption in the uterus. These observations were further investigated by Cohlan.[48] He administered *per os* to pregnant female rats 35,000 IU vitamin A daily from the 2nd, 3rd or 4th to the 16th day after coitus and found 'that the administration of excessive amounts of vitamin A to pregnant rats produces a diminished litter rate and characteristic malformations among the surviving young.' The anomalies included extrusion of the brain, macroglossia, harelip, cleft palate and eye defects. The cranial deformity was consistent.

Vitamin B_2 (Riboflavin). Warkany *et al.*[5, 9] and Giroud *et al.*[11] describe malformations in the offspring of mother rats on diets deficient in riboflavin during pregnancy. The former workers found that the malformations in rats were not determined before the 13th day of gestation and that, up to that time, liver supplements to the maternal diet prevented their occurrence. However, once the malformations were established supplements to the maternal diet might prevent abortions but would not correct the malformations.

Vitamin B_{12}. Hogan and Richardson (quoted by Giroud and le Febvres-Boisselot [12]) produced experimental hydrocephalus in rats which was in a measure attributable to vitamin B_{12} and folic-acid deficiency. Lepskovsky and his co-workers [13] studied the effects of vitamin-B_{12} deficiency on viability, growth and reproduction in 3 successive litters of rats. There was high mortality in the young 2 to 3 weeks after weaning, even when they were nursed by normal mothers. Severe intra-uterine injury was shown by third-litter rats. The abnormalities became progressively severe in successive litters. Olcese *et al.*[16] describe congenital anomalies in chicks due to vitamin-B_{12} deficiency. In eggs laid by hens on a B_{12}-deficient diet there was a high mortality in the embryos at the 16th to 18th day of incubation. The most characteristic deformity was 'spindly legs' (myo-atrophy of the legs), which also had a haemorrhagic appearance. Of unusual frequency was the abnormal position of 'head between the thighs'. O'Dell *et al.*[17] produced offspring with numerous physical abnormalities by placing female rats on B_{12}-deficient diets. Hydrocephalus occurred in approximately 23% of the foetuses. There was also a high mortality in the young rats. Weekly injections of 1.0 μg. B_{12} into female rats, before and during gestation, prevented hydrocephalus in the young. It appeared that damage to the foetus of mothers on B_{12}-deficient diets occurs at the 12th to 14th day of gestation.

Folic Acid. According to Nelson and Evans (*quoted by* Giroud and le Febvres-Boisse'ot [12]) folic-acid deficiency in the pregnant rat causes absorption of some embryos and diminution in the sizes of the survivors. As folic acid is synthesized in the rat's intestine, deficiency of this vitamin in the rat can be produced only by preventing such synthesis. This was done by succinylsulphathiazole in the pregnant rat. The resulting deformities in the foetuses included coloboma, harelip, big facial clefts, nasal atrophy, coelosomia and ectocardia.

Riboflavin. Piccioni *et al.*[18] kept mother rats on a riboflavin-deficient diet from 40 days before pregnancy and throughout the period of pregnancy. Only 30 out of 53 rats were born alive and the progeny showed the following malformations: shortness or absence of limbs, syndactylia, cleft palate, shortness of the mandibula, and protrusion of the tongue. A few mothers died towards the end of the gestation period.

Pregnancy and lactation are associated with increased requirements for most nutrients including riboflavin.[19] Further, severe injury, illness or burns may increase riboflavin requirements 5–10 times.[20]

Pantothenic Acid. Le Febvres-Boisselot [21] determined the effect of pantothenic-acid deficiency on the offspring of rats. Malformations occurred in 94% of the offspring of the experimental rats on a synthetic diet deficient in pantothenic acid. The malformations included exencephaly, pseudoencephaly, anophthalmus and oedema and haemorrhage of the extremities. The parent rats showed no signs of any of these malformations.

Other Deficiencies. Polman [22] studied congenital malformations in certain areas in Holland. The incidence in 2 groups of families was very high. He was unable to state whether iodine-deficiency played any rôle in the causation of the malformations. In the Union of South Africa, as well as in many other countries, many thousands of people have been examined in endemic goitre areas and no evidence of an exceptionally high incidence in foetal deaths and/or malformations has been recorded. However, in Switzerland deaf-mutism and retardation of mental development appear to be prevalent in endemic goitre areas. It is, of course, well known that hypothyroidism (iodine deficiency) is associated with retardation in mental and physical development.

According to Bennetts and Chapman [52] and Smith [53] deficiencies in certain minerals (copper and iodine) may cause congenital abnormalities.

(B) TOXIC AGENTS, INCLUDING MATERNAL DISEASES AND CONDITIONS WHICH INDUCE ANOXIA IN THE FOETUS

From the investigations conducted by Ingalls *et al.*[23] and observations made by Olim and Turner [24] it is clear that any disease or condition which induces a reduced supply of oxygen to the foetus may cause malformations. The type and extent of the deformity depend on the stage of development of the foetus, the period during which the insult acts and the intensity of the action. Any type of deformity can be caused *in utero* by the action of various insults. Ingalls and his co-workers submitted pregnant mice at various stages of pregnancy to rarefied atmospheres and found that maternal anoxia may result in foetal death or congenital malformations. They say, 'The effect on the conceptus varies with the degree of maternal placental anoxia and the stage of gestation at which the anoxia insult occurs'. They conclude that rapidly differentiating tissues are more vulnerable to anoxia than resting or fully-differentiated cells. The method they prescribe for the evaluation of rôles played by teratogenic agents, the foetal host, and the maternal placenta, in the production of acquired congenital anomalies is of great value.

Olim and Turner [24] describe a case of a young mother with a congenital cardiac defect who gave birth to two anencephalic foetuses. Anencephaly was caused by the low arterial oxygen saturation which interfered with the nutrition of the foetus during the cephalic phase of development. Each of the two infants were born at approximately 6 months' gestation. After correction of the cardiac defect by the Blalock operation there was material improvement in arterial oxygenation, so that the patient could then carry on her normal activities without cyanosis or dyspnoea, and she then gave birth to a normal child.

Mitotic Poisons. The following chemicals are known to disturb processes of mitosis: colchicin, selenium, nitrites, 'miracil' (thioxanthone compound), urethane, trypan blue, members of the quinone group, nitrogen mustards, arsenic, sodium cyanate, potassium sulphocyanate, acridine dyes, benzene, phenols, several steroids, γ-hexachlorocyclohexane, 1-amino-acenophthen and derivatives,[26] adrenalin,[27] mustard gas,[28] compounds related to mustard gas,[29] stilbamin derivatives,[32] and phenylcinnamic acid nitrites.[33]

Chemical substances which have produced malformations in animals are colchicin, insulin, selenium, urethane, boracic acid, pilocarpine, mustard gas and trypan blue (Gillman *et al.*,[38, 39] Grebe and Windorfer [15]). Dammers

and Frens [25] describe abnormalities of the penis in young rats resulting from the administration of iodocasein. Staemmler [31] states that in animals it has been proved that alcohol causes changes in mutation and damages sperm cells and that we must accept the same possibility in man. Gillman *et al.*[38, 39] found that a single injection of 1 ml. of 1% aqueous solution of trypan blue given to female rats during the early stages of pregnancy could result in gross malformations in their offspring whereas an injection given before as well as another during pregnancy greatly enhanced the incidence of gross defects in the new-born. It is of importance to note that congenital malformations are rare in the offspring of mothers given a single injection of trypan blue before pregnancy but that metabolic disorders, such as jaundice, occurred in 1.8% of the newborn. The following congenital defects in the offspring of mother rats injected with trypan blue were recorded by Gillman and his co-workers: Eye, ear and tail defects, hydrocephalus, hip dislocations, spina bifida, umbilical hernia, cleft palate, harelip, deformed hindlimbs, club foot, skull defects, meningocoele, imperforate anus, microcephalus, and craniaschisis. The total percentage of abnormality was 32.[4]

Fraser *et al.* produced cleft palates in 79% of the offspring of pregnant mother mice injected with cortisone.[30] An important feature of their results is the fact that cleft palate also occurred in foetuses when treatment of the mother mice with cortisone was started after the 12th day of gestation, when the nasomaxillary fissure is said to have closed. There is thus a possibility of cleft palate due to degenerative changes. The quantities of cortisone administered to the pregnant mice were large. Nevertheless, the results are of great significance to man as cortisone is used in treatment on a large and growing scale.

Kimball [34] and Chury [36] refer to the induction of mutations in animals by radiation, while Loveless [35] discusses the quantitative aspects of the chemistry and biology of radiomimetic (mutagenic) substances.

Lettré *et al.*[33, 37] investigated synergists of mitosis. They [33] found that phlorrhizin stimulated non-mitotic quantities of colchicin and N-methylcolchicamide into mitotic action. They suggest that a possible explanation of this phenomenon is the inhibition of phosphorylation processes by phlorrhizin.

The observations made by Grebe and Windorfer [15] are of very great significance as indicating that chemical abortifacients and chemical contraceptives are possible causes of congenital malformations. They describe a case where a mother had given birth to a deformed child after she had used contraceptive tablets. The child showed anophthalmia (left), microphthalmia (right) with defective eye-lid formation, harelip, cleft palate, microcephalus, arhinencephalia (left), absence of nervus and tractus opticus, congenital heart defect with subaortal septum defect and common atrium, stenosis of aorta isthmus, and absence of the anal orifice. It died 3 months after having been admitted to hospital.

Hone and Magarey [44] described a case of cretinism caused by the administration of methylthiouracil to the mother during pregnancy. They recommend that in cases where it is necessary to administer this drug during pregnancy, administration should always be discontinued at least 3 weeks before term. Poate's [45] experience has been that if the smallest effective doses of 'thio' drugs in thyrotoxicosis is given, no harm is done to the foetus. He states that 'colloid does not appear in the gland of the human foetus until about the 6th month, so that the possible danger-period to the foetus is from this time until full term, when thyroxine and iodine are necessary'. He rightly recommends that it is wise to stop administration of antithyroid drugs in the last 6 to 10 weeks of pregnancy and to give small doses of Lugol's solution (3 drops twice daily) and to continue this for 3 months after parturition. Bickenbach *et al.*[46] administered methylthiouracil orally to pregnant rats. The young of those mothers which received the drug during pregnancy and lactation, or during lactation only, showed all the signs of cretinism. Szontagh and Lichner [47] found that methyl- and propylthiouracil decrease the number of oestrus days in young healthy rats. Histologically the ovaries showed marked luteinization and few follicles.

DISCUSSION

Practical experience and the results of investigations into the effects of vitamin deficiencies, anoxia, maternal diseases and mitotic poisons on animal and human embryos turn our minds to the practical application of the knowledge acquired. Heredity can no longer be regarded as the sole cause of teratism. As a matter of fact, it appears that the majority of foetal deaths and malformations are due to causes other than heredity; consequently efforts on the right lines can and will prevent many of the common congenital malformations. It appears that not only vitamin deficiencies (A, B_2, B_{12}, folic acid, riboflavin and pantothenic acid) before and during pregnancy but also conditions (haemorrhage, maternal heart defects, etc.) or poisons which induce anoxia in the foetus, maternal diseases (rubella), or chemical substances which disturb mitotic processes, may cause different types of congenital malformations in man and animal. The nature of the foetal abnormalities depends on (1) the type of insult, (2) the degree of the insult, (3) the length of the period of its action, and (4) the stage of development of the foetus. The younger the foetus and the more active the development of the tissue is at the time the insult acts, the greater and more extensive the abnormalities will be. It should be noted how prevalent harelip and cleft palate are among congenital abnormalities irrespective of the cause of the deformities.

Gillman *et al.* have shown that we should think not only of physical abnormalities but also of metabolic disturbances (e.g. jaundice) in the foetus as a result of insults during pregnancy.

From the work of Fraser and his associates it appears possible that some agents (cortisone) may not only prevent the full development of certain foetal tissues but may even destroy tissues which have already been formed.

Ingall's publications [41, 42] have thrown much light on the possible *in utero* causes of mongolism. He suggests that we centre our attention on the 6th to 9th weeks of foetal life in our search for possible etiologic agents (insults). He [42] states, 'While the causative agents of mongolism are relatively numerous, the causative mechanisms are few in number and operate at about the

8th week of fetal life. Causative agents include hemorrhage, threatened abortion, pathologic abnormalities of the uterus and certain acute intercurrent infections. Experimental and clinical evidence suggests that lack of oxygen to the fetus may be an important mechanism, with temporary starvation and the accumulation of toxic metabolites to be evaluated'.

De Rudder [42] states that at present the general view is that the syndrome of mongolism is caused by damage to the plasma of the ovum which was fertilized. He refers to the observation made by Klebanow that in children of mothers who have during pregnancy experienced extreme physical, spiritual and mental stress the percentage of mongolism is very high. According to de Rudder the often suspected hereditary nature of mongolism has not been proved.

In all cases of congenital physical and mental defects due consideration should be given to the possible rôle played by *chemical* contraceptives, abortifacients, and drugs used during pregnancy.

From the literature it is clear that attention should also be paid to synergists of mitotic poisons, for the former enhance the harmful effects of the latter.

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ABSTRACT

The First Day of Life. Editorial (1953): J. Amer. Med. Assoc., **151,** 477.

The decrease in the death rate during the first year of life has not been accompanied by a corresponding decrease in the rate during the first 24 hours. Only through medical skill will it be possible to further reduce mortality of the newborn.

While prematurity, asphyxia, atelectasis, birth injuries and congenital malformations contribute to neonatal death, other factors increase the chance of survival, e.g. good maternal care, elimination of infections, adequate nutrition, and skilful delivery with a minimum of interference and untoward effects from analgesia and anaesthesia.

To establish extra-uterine respiration, when postural drainage and suction do not suffice, direct laryngoscopy and tracheal suction may save life, and so may mouth-to-mouth insufflation.

Initial examination of the newborn is important, with careful search for bleeding points, jaundice, heart murmurs and lung lesions, and for evidence of congenital malformations or birth trauma.

A thorough examination of the anterior fontanelles should be made within 24 hours of birth and of the eyes, ears, nose, lips and palate for congenital defects. If an anomaly is found, others should be sought. Disparity in respiratory excursion and cardiac shift should encourage X-ray studies. If anaemia, jaundice or infection is present, haematologic studies should be carried out. Infants born of diabetic mothers and those delivered post-maturely or by Caesarean section should be given particular attention.

South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

VAN DIE REDAKSIE

LOODVERGIFTIGING BY KINDERS

Op die gebied van openbare gesondheid speel die vergiftigende eienskappe van lood 'n groot rol. In die industrie het dit in die verlede hoë siekte- en sterftesyfers veroorsaak. Op plekke waar die watervoorraad vir publieke gebruik loodoplossend was is grootskaalse loodvergiftiging deur die gebruik van loodwaterpype en ander loodtoebehore veroorsaak.

In die jongste tyd is die loodvergiftiging van kinders, vernaamlik in die ouderdomsgroep 1 tot 2 jaar, onrusbarend. Die verf word van speelgoed, speelhokke, wiegies, kinderbedjies, vensterbanke, stoeptralies gesuig of deur vuil handjies opgeneem. Vergiftiging is ook veroorsaak deur geverfde houtwerk wat afskilfer, en is waargeneem in kinders wat aan 'n loodtepelskerm suig.[1] Dit is selfs in babatjies bespeur wat melk gedrink het afkomstig van koeie wat gewei het op gras voorheen met loodarsenaat bespuit.[2]

Enige maande gelede [3] is loodvergiftiging van kinders in hierdie Tydskrif bespreek maar omdat so min mense in Suid-Afrika van hierdie gevaar bewus is, haal ons die onderwerp weer aan om die feit te beklemtoon dat die vergiftiging van kinders op hierdie manier nie seldsaam of onwaarskynlik is nie.

Volgens een groep skrywers [4] is loodvergifting een van die algemeenste oorsake in die Verenigde State van Amerika van kindersterftes aan vergiftiging. Op grond van eie ervaring en 'n studie van die literatuur kom hulle tot die gevolgtrekking dat baie van die onverklaarbare senuwee-aandoenings en stuiptrekkings van onbepaalde etiologie te wyte kan wees aan loodvergiftiging wat nie in die kinders herken word nie. Kinders is besonder vatbaar vir loodvergiftiging en hul simptome verskil grootliks van die van volwassenes. Stoornisse van die sentrale senuweestelsel of loodvergiftiging van die harsings kom selde voor in volwassenes maar is algemeen in kinders; loodverlamming, loodmerke op die tandvleis en koliek is gewoonlik in kinders afwesig.

Gedurende 'n tydperk van 2 jaar, is 23 gevalle van loodvergiftiging tot 'n kinderhospitaal in Toronto toegelaat.[5] Veertien gevalle van kroniese loodvergiftiging in 'n ander reeks kinders [6] tussen die ouderdomme 14 maande en 3½ jaar was hoofsaaklik te wyte aan verf afkomstig van vensterbanke en mure; 10 gevalle het definitiewe simptome van loodvergiftiging van die harsings getoon en dit was uiters moeilik om 'n differensiaaldiagnose te maak wanneer harsing- en rugmurgvliestering ter sprake was. Kenmerke van loodvergiftiging van die harsings behels sulke simptome soos prikkelbaarheid, slaperigheid, stuiptrekkings, uitpeulende fontanelle, papillodeem en verhoogde druk van die harsings- en rugmurgvliesvloeistof.[7] Radiografie van die bene is van belang met diagnose; die eindpunte van die jong, vinniggroeiende bene toon digte lyne aan naby die epifeses en die groeiende rante van die bene. Hierdie lyne is nie patognomonies nie.[8] Noodlottige

EDITORIAL

LEAD POISONING IN CHILDREN

The poisonous qualities of lead are important in several public-health relationships. In industry it has in the past caused vast amounts of mortality and disease. The use of lead for water pipes and other fittings has caused extensive outbreaks of lead poisoning in places where plumbo-solvent water was used for public supplies.

More recently accidental poisoning in children has given concern, especially in children one or two years old, brought in contact with lead paint on toys, cribs, cots, play-pens, window sills and verandah railings. The paint is ingested by sucking or from soiled hands. Poisoning has also been caused by paint flakes falling from woodwork. It has been recorded in children who suck a nipple-shield made of lead.[1] Poisoning has even occurred in infants from the drinking of milk from cows that had eaten weeds previously sprayed with lead arsenate.[2]

The subject of lead poisoning caused in infants by paint was considered in this Journal some months ago,[3] but as few people in South Africa seem to be impressed with the danger we return to the subject in order to emphasize the fact that lead poisoning in children is not rare or unlikely.

According to one group of authors [4] lead poisoning ranks in the United States as one of the commonest causes of fatal poisoning in children. From a study of the literature and from their own experience of many cases they conclude that unrecognized lead poisoning in children may explain many obscure nervous conditions and convulsions of undetermined aetiology. Children are particularly susceptible to lead poisoning and in them the symptoms differ considerably from those in adults. Central nervous system involvement or encephalopathy, rarely seen in adults, is common in children; lead palsy, lead line on the gums and colic are usually absent.

In 2 years, 23 cases of lead poisoning were admitted to a children's hospital in Toronto.[5] Fourteen cases of chronic lead poisoning which occurred in another series [6] in children 14 months to 3½ years old resulted mainly from the ingestion of paint from window sills and walls; 10 cases had definite evidence of lead encephalopathy, presenting great difficulty in differential diagnosis when there was a question of meningeal tuberculosis. The features of lead encephalopathy include such symptoms as irritibility, drowsiness, convulsions, bulging fontanelle, papilloedema, and raised cerebrospinal fluid pressure.[7] Radiography of the bones is important in diagnosis; the extremities of the young rapidly-growing bones show dense lines near the epiphyses and growing edges of the bones. These lines are not pathognomonic.[8] Fatal cases

gevalle van loodvergiftiging van die harsings wanneer so 'n diagnose nie klinies verdag was nie kon deur die patoloog gediagnoseer word van tipiese makroskopiese veranderings in die organe en van kwantitatiewe toetse vir lood.[9]

Die prognose vir loodvergiftiging in kinders is ernstig. Hoe jonger die kind hoe hoër die sterftesyfer. Meer besonderhede van die simptomeleer, diagnose en behandeling word in die naslaanlys vervat wat in hierdie artikel aangegee word. In besonder moet die aandag bepaal word op die gebruik van die chelaterende middel *disodium calcium ethylenediamine tetra-acetic acid* (EDTA) wat 'n vaste samestelling met lood vorm wat baie vinnig in die urien uitgeskei word. Dit is meer doeltreffend om hierdie middel binneaars as mondeling toe te dien en dit word goed geduld.[10] *Dimercaprol* is van geen waarde met die behandeling van loodvergiftiging nie. Loodvergiftiging van kinders kan vermy word deur ouers, vaders sowel as moeders, te leer om verfstowwe wat lood bevat vir kinderspeelgoed en kateltjies te vermy. Ouers behoort oor die gevare van loodvergiftiging ingelig te word. Vervaardigers van speelgoed kan hierdie gevaar uitskakel. In baie lande word verfstowwe wat lood bevat nie vir die vervaardiging van wiegies en speelgoed gebruik nie. Titanium, antimoon en sinkoksiedes het wit lood in baie glansverfstowwe en emaljes vervang, alhoewel lood nog in sommige verfstowwe, vernaamlik die geel en groen soorte, gebruik word.[8] Die maatreëls wat in ander lande aangewend word om vergiftiging te verhoed word genoem in een van die naslaanartikels waarna ons verwys.[4]

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9. Finkelstein, W. E. (1952): J. Mt. Sinai Hosp., **19,** 352.
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of lead encephalopathy in which the diagnosis was unsuspected clinically may be diagnosed by the pathologist from typical macroscopic changes in the organs and from quantitative estimations of lead.[9]

Lead poisoning in children carries a grave prognosis. The younger the child the higher the mortality. Further details of the symptomatology, diagnosis, and treatment are to be found in the references given in this article. Attention may in particular be drawn to the use of the chelating agent disodium calcium ethylenediamine tetra-acetic acid (EDTA), which forms a stable complex with lead which is rapidly excreted in the urine. This drug is more effective intravenously than orally and is well tolerated.[10] Dimercaprol is not of value in lead poisoning.

The prevention of lead poisoning in children will depend on the education of parents, fathers as well as mothers, in the use of non-lead paints on children's toys, beds, etc. The dangers of lead poisoning should be brought to their notice. Manufacturers of toys can obviate the danger. In many countries lead-containing paint is not used in the manufacture of toys and cribs. Titanium, antimony and zinc oxides have replaced white lead in many gloss paints and enamels, although lead is still used in some, especially in yellow and green varieties.[8] The measures used to prevent poisoning in other countries are reviewed in one of the articles we cite.[4]

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ABSTRACT

Immunity of the Newborn. Murray, J. and Calman, R. M. (1953): Brit. Med. J., **1,** 13.

Most of the work indicating that placental cord blood contains more antibodies than maternal blood has been done with diphtherial and staphylococcal antibodies. Working at the Bernhard Baron Memorial Research Laboratories, Queen Charlotte's Maternity Hospital, London, the authors have estimated blood levels in placental and cord blood employing the streptococcus and utilizing antistreptolysin. Materials, methods and techniques are described in detail. The authors ruled out to their satisfaction the possible errors resulting from the presence of inhibitory serum factors such as cholesterol and ascorbic acid.

In 33 of 46 cases the cord blood level was at least twice that of the maternal blood. In 1 instance both levels were too low to indicate a significant difference. In the remaining 12 cases differences were not pronounced. However, in 8 of these the antistreptolysin titre was higher in cord blood than in maternal blood and in 3 cases the levels were approximately equal. In 1 case the maternal level was higher. In 1 case both levels were too low for the test.

The results lend additional support to previous conclusions of the authors that simple filtration does not account for the passage of antibodies between maternal and foetal blood.

THE DIAGNOSIS AND TREATMENT OF MALE INFERTILITY

WITH SPECIAL REFERENCE TO ITS ENDOCRINOLOGIC ASPECTS*

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Whereas the study of other endocrine glands has been limited to the last century, knowledge of the endocrine function of the testis is nearly as old as medicine itself. The practice of castration, both of men and of animals, shows that it was common knowledge that the presence of the testis is necessary for the appearance of certain growth and behaviour characteristics. The wider implications of this information seem to have been missed, however, and further study of testicular endocrinology was postponed until the last century, and in particular until investigation of male sterility was undertaken.

The growth of knowledge of male infertility was also delayed. Polygamous societies were well aware that male potence† is not synonymous with fertility—as any reader of the unexpurgated *Arabian Nights* may discover—but a few millenia of monogamy made it easy to ignore this blow to masculine pride. Although Sims [1] stressed the importance of demonstrating spermatozoa in semen as far back as 1868, the systematic investigation of the husband in a sterile union was deferred until about 25 years ago.

Strictly speaking, an infertile union is one which produces no pregnancies within the reproductive life of the couple. In practice, however, for treatment of infertility to be possible, one must set a time limit and regard all couples desiring children but failing to produce a pregnancy within this period as at least relatively infertile. Two years is a common choice, but Bender [2] recommends it be reduced to 12 months of 'uncontracepted married life'. This naturally produces a high rate of self-curing 'infertility', which Bender encountered in nearly 30% of his cases.

Using these practical criteria (of 1 or 2 years involuntary infertility) most workers find that about 10% of all marriages are infertile, and that some degree of male responsibility can be discovered in half of these. In under 10% of cases (7% Bishop [3]) the male is azoospermic, i.e. in nearly 1% of all marriages.

What proportion of these infertile husbands are legitimately to be regarded as endocrinologic in aetiology or in treatment is not known; at present we must admit that the proportion is likely to be small, but will probably increase.

REQUIREMENTS FOR MALE FERTILITY

The following represents the simplified chain of causality involved in male fertility:

1. A normal adult endocrine system, involving, in particular, normal hypothalamus, anterior pituitary, adrenal and thyroid glands, together with normal liver function.

2. Testes capable of responding adequately to endocrine stimuli by production of spermatozoa, androgens and oestrogens. Spermatogenic response, for example, requires testicular temperatures several degrees below body temperature, and the slight rise of temperature involved in ectopia or in fevers [4] abolishes this capacity.

3. Normal ripening of the spermatozoa in the epididymides and a normal rate of conduction through epididymides, vasa deferentia and ejaculatory ducts. Interference in conduction, whether it be by congenital aplasia of ducts,[5] or post-inflammatory obstruction, results in obstructive azoospermias.

4. Normal contributions of the seminal vesicles and prostate glands.

5. Potence, i.e. the masculine ability for normal sexual intercourse, involves first, erection and orgasm, and secondly, ejaculation. This complicated function demands the co-operation of both sympathetic and parasympathetic nervous systems,[6] as well as being considerably influenced by the higher centres. Erection and orgasm require in addition to local anatomical features, an intact pelvic parasympathetic nervous system, i.e. sacral nerves 2, 3 and 4. Cauda equina lesions may cause impotence of this type. Ejaculation, on the other hand, depends on a functioning sympathetic system. The sympathetic ganglia involved are sometimes L1 but are more generally higher than this (T12), and the pathway involves the presacral nerves. Consequently extensive sympathectomy such as is undertaken for hypertension may produce sterility by abolishing the ejaculatory ability, whereas lumbar sympathectomy for vascular lesions in the legs is generally innocuous in this respect.[7]

6. Finally, insemination must coincide with ovulation—a matter which normally may be left to chance, but which sometimes requires special instructions.

Associated with each stage in this process is a particular group of disorders giving rise to absolute or relative infertility: hence precise diagnosis is necessary for treatment to be effective. Systematic investigation is essential, and it is noteworthy that progress both in diagnosis and therapeutics has depended very largely on the invention of new tests, notably testicular biopsy and estimation of urinary gonadotrophin excretion.

TESTS OF VALUE IN THE INVESTIGATION OF MALE FERTILITY

A. *Direct Tests*

1. *Gonadotrophin Production.* The urinary level of follicle-stimulating hormone (F.S.H.) enables one to distinguish between 2 main classes of testicular failure:

* Paper presented in May 1953 at the Endocrinology Conference of the Students' Medical Council, University of the Witwatersrand.

† In conformity with the term 'impotence', and on account of the number of other medical meanings of 'potency', the word 'potence' is used for 'potentia coeundi' in this paper.

(i) testicular failure secondary to pituitary hypofunction, where the gonadotrophin excretion is low, and

(ii) testicular failure where the pituitary is functionally normal. The gonadotrophin level in this group is normal or may be considerably raised as a result of lack of pituitary inhibition by testicular hormones (androgen, oestrogen and, possibly, 'inhibin').

The test employed is a modified Ascheim-Zondek, where the effect of F.S.H. on the Graafian follicles is measured by the influence of follicular secretion on uterine weight. The test may be done either on a 16- (or 24-) hour specimen (dialysis method) or on a 90-minute specimen (non-dialysis method). The results are expressed as 24-hour excretion. By the dialysis method, used in recognizing low gonadotrophin levels, the normal range is 6.5–26 mouse-units per day. With the non-dialysis method, which is more suitable for recognizing high levels, figures of 96 mouse-units and higher are regarded as abnormal. Interstitial-cell-stimulating hormone (I.C.S.H.) can be estimated by the Xenopus pregnancy test, but this is too crude for use in male infertility.

2. *Androgen Production.* 17-ketosteroids are believed to be excretory transformation products from certain adrenal and testicular steroid hormones. Not all androgens are excreted thus, however, nor are all 17-ketosteroids in the urine derived from androgens, and their level is only a rough guide to the testicular function. The normal urinary figures are 7–25 mg. per day and decreased excretion may occur in malnutrition, infections and liver disease. As one might expect, low levels are found in eunuchoidism, especially in that due to hypopituitarism and castration.

3. *Testicular biopsy* is even more useful in male sterility than endometrial biopsy in female sterility, for it indicates both hormonal and gametogenic states. It is essential for differentiation of different forms of azoospermia, oligozoospermia and eunuchoidism, and the information it provides makes rational endocrinologic treatment possible.

The handling and fixation of the biopsy specimen are critical. Slight pressure with forceps causes extensive exfoliation of seminiferous epithelium, and rupture of nuclei. Fixation in 10% formalin causes shrinkage during paraffin embedding, and nuclear details may be obliterated. For routine use, Bouin's solution is recommended—saturated picric acid 75 vol., formalin formaldehyde 40 vols. % 25 vols., glacial acetic acid 5 vols.

4. *Hyaluronidase Content of Semen.* The enzyme hyaluronidase is carried on the spermatozoa, and its concentration in semen can be closely correlated with the sperm count.[8] Most workers claim that it is absent from prepubertal and undescended testes and from azoospermic ejaculates. Michelson and Koets,[9] however, claim to have found hyaluronidase in the semen of 17 out of 31 azoospermic ejaculates, and regarded its presence as proof of patency of the ductal system. Nevertheless, in 5 of their cases with hyaluronidase that were biopsied, 3 showed spermatozoa in the testis cases (3, 7 and 13), i.e. obstructive azoospermia. Michelson and Koet's figures therefore do not support their own hypothesis, and there seems no justification at present for assays of seminal hyaluronidase.

5. *Intravasal Injection of Dye.* Since obstruction to the escape of spermatozoa can occur in the epididymis as well as in the vas or ejaculatory ducts, the patency of the vas can be checked by an intravasal injection of a dilute non-toxic dye such a methylene blue or mercurochrome. Escape of dye from the urethra or its appearance in the urine is easy to recognize. Use of a radio-opaque material, e.g. equal parts of 20% uroselectan and argyrol, followed by radiography, demonstrates more precisely the site of blockage.[10]

6. *Microscopical Examination of Semen.* This investigation is the simplest and most important test for male fertility. The standards have recently been revised, and it is now recognized that pregnancies may occur with counts much lower than the previous limits of 60 million per ml. and in fact have been reported with counts of less than 1 million per ml.[2] The need for normal motility and normal morphology is confirmed, but there is less assurance as regards the effects of relative shortcomings in any single feature. Certainly no one defect can be correlated with an increased tendency to miscarry,[11] and although Moench has claimed that abnormal head morphology might be responsible for abnormal conceptions, evidence for this is lacking.[10, 11]

Table I gives, in round figures, current opinion regarding semen values in fully fertile men. Figures lower than the suggested minimum for full fertility represent subfertility rather than absolute infertility.[12, 13, 14]

TABLE I

	Average	*Range*	*Suggested minimum*
Ejaculate volume in ml. ...	3.5	1– 11	1.0
Density of spermatozoa in millions per ml.	140	30–700	20
Total count in millions ...	400	70–800	30
Percentage motile in first hour	80	50– 85	50
Percentage with abnormal head morphology	10	6– 15	Should not exceed 25

As has already been indicated, minimum standards have been steadily falling, especially since it is recognized that the degree of fertility demanded of a husband is much less exacting than that required of a pedigree bull, where every service must be paid for.

In a study of 1,600 men of proven fertility McLeod and Gold [14] have demonstrated that they could find no relationship between ease of conception and ejaculate volume or sperm morphology. Sperm counts below 20 million per ml. seem to delay conception, but above this level there is no hint of a relationship between sperm count and ease of conception. Conception occurred soonest when the percentage of active cells was above 60%. Quality of motility seemed to be the most significant single feature.

The same authors [15] found that the semen from husbands of proved fertility tended to be better in all respects as compared with that from husbands of infertile marriages, and from this comparison they concluded that all aspects seem to be important.

The outlook is far from hopeless in male seminal deficiencies, and relative infertility in the male appears much less gloomy prognostically than does relative infertility in the female—as one might expect in view of the limited nature of his role in propagation.

7. *Seminal vesicular tests.* Mann [17] has demonstrated that the vesicular secretion is unique in containing large amounts of fructose. Normal semen may thus have a fructose content (estimated as glucose) as high as 640 mg.% (mean 287).[18] In obstruction of the ejaculatory ducts this falls to zero [19]—a useful test for patency. Lundquist [20, 21] has shown that phosphoryl choline is another characteristic vesicular product. Using fructose content and fractionated ejaculates it has been shown that haemospermia is generally vesicular in origin.[22] The specific secretions of the seminal vesicles are dependent on the presence of male sex hormone, and the 'fructose' test is a very sensitive indicator of its presence.[23a, 23b]

8. *Prostatic Secretion Tests.* Examination of the prostatic secretion is seldom necessary, except microscopical examination for the presence of pus and organisms. Adult prostatic fluid characteristically contains high levels of acid phosphatase (15,000 King and Armstrong units per ml.)[24, 21] citric acid (0.48–7 g.%), and fibrinolytic enzyme.[25] The levels of these substances may possibly be employed as tests of prostatic function since prostatic fluid normally represents 12.8–32.5% of the whole ejaculate.[21] It will be borne in mind, however, that the levels of these secretory products will depend on the presence of circulating androgens, as Hansen has demonstrated.[24]

9. *Tests for Potence.* The history is usually reliable in cases of sterility, but a post-coital examination of the vaginal pool and the cervical mucus (Sim's test) is the only laboratory proof of potence and the invasive power of spermatozoa. Its practical value is slight.

10. *Tests for Coincidence with Ovulation.* A chart of the wife's basal temperature with menstrual and coital data is a useful indication of true infertility, as distinct from artificial infertility owing to non-coincidence with ovulation.

In the absence of more exact demonstration of coincidence, frequency of random intercourse is a useful guide to the chance of conception, and has been shown to be directly related to ease of conception.[14] This may seem remarkable as semen-quality improves with increasing periods of continence up to 10 days [16]: evidently the deterioration in semen-quality associated with more frequent intercourse is far less significant than the increased opportunity for coincidence with ovulation.

B. *Therapeutic Tests*

Since potent hormone preparations are now available, it is possible to use therapeutic tests of endocrine deficiency.

1. *Response to Chorionic Gonadotrophin (I.C.S.H.).* Heller and Nelson [26] regard failure of a hypogonadal patient to respond clinically to 750 i.u. of chorionic gonadotrophin intramuscularly twice daily for 3 weeks as proof that the primary defect lies in the testis.

2. *Response to Testosterone.* Testosterone propionate will counteract the symptoms of eunuchoidism, but such treatment is not to be undertaken lightly since, as Heller and Nelson [26] have pointed out, testosterone may cause sufficient testicular and pituitary suppression to prevent subsequent stimulation of the testis. Doses of 25 mg. of testosterone propionate intramuscularly daily for 2 weeks will distinguish cases of Leydig-cell failure, e.g. the male climacteric, from psychogenic disturbances.[27] In Leydig-cell failure, gradual alleviation of symptoms occurs and continues for a day or two after injections cease, relapsing from the 3rd to the 7th day after cessation. If Leydig cells are normal no response occurs or, in co-operative neurotics, a sudden improvement may occur at the beginning with immediate relapse at the end of treatment.

PITUITARY-GONAD RELATIONSHIPS

From the endocrinological point of view, the most susceptible functions are the pituitary-gonadal relationships and maintenance of potence.

The anterior pituitary contains certain chromophil cells which have long been classified as eosinophil (alpha) and basiphil (beta and delta). As regards the pituitary hormones that concern sex, there is evidence that the follicle-stimulating hormone (F.S.H. or Prolan A) is produced by delta cells,[28, 29] and the interstitial cell-stimulating hormone (I.C.S.H., luteinizing hormone, L.H. or Prolan B) is secreted by special alpha cells—the 'carminophiles' [28] and probably also the delta cells. Corticotrophin (A.C.T.H.) is probably secreted by beta cells.[29] This hormone stimulates the adrenal cortex to liberate androgens and is thus responsible for some of the 17-ketosteroids found in urine.

More direct evidence is provided by Pearse,[30] using a stain for glycoprotein. This in the anterior pituitary distinguishes a group of 'mucoid cells', comprising the basiphils and some of the chromophobes which, he suggests, very reasonably, are the cells responsible for the carbohydrate-containing hormones (F.S.H., I.C.S.H. and thyrotrophic hormone).

Lesions of the hypophyseal stalk cause reduction or complete disappearance of granulated pituitary cells, and the testicular effect is similar to that of hypophysectomy.[31] This author states that lesions of the hypothalamus itself occasionally caused specific testicular changes, viz. a degeneration confined to spermatozoa and maturing spermatids, distinguishable from purely hypopituitary effects.

I.C.S.H. acts on the interstitium of the testis inducing the differentiation of adult Leydig cells and maintaining their secretion of androgens, and possibly oestrogen as well. F.S.H. acts on seminiferous tubules, both on Sertoli cells which change to the adult form and apparently secrete oestrogen, and on spermatogenic cells, which are induced to differentiate. Spermatogonial mitoses seem to be independent of gonadotrophic stimulation.

In addition to its effect on secondary sex-character, androgen has been shown in experimental animals to exert a local testicular effect, independent of gonadotrophins, and according to Gaarenstroom and de Jongh,[32] appears to preserve the stratification of the germinal epithelium. It also inhibits secretion of gonadotrophins.

Oestrogen has not been shown to exert any local testicular effect, but inhibits gonadotrophin production. The liver appears to be of considerable importance in the metabolism of these steroids, and hence it is not surprising that in severe liver disease gross disturbance of spermatogenesis results; presumably impaired steroid metabolism lead to high androgen and oestrogen levels, which cause depression of F.S.H. and I.C.S.H. production, and spermatogenic arrest.

SYNDROMES

TABLE 2

A. *Tubulo-Interstitial Hypogonadism.*
1. Hypopituitary hypogonadism
   a. Panhypopituitary hypogonadism
      i. Juvenile panhypopituitarism
      ii. Adult panhypopituitarism
   b. Hypogonadotrophic hypogonadism
      i. Primary juvenile hypogonadotrophic hypogonadism
      ii. Primary post-pubertal hypogonadotrophic hypogonadism
2. Hypogonadism associated with hepatic deficiency
3. Total hypogonadism due primarily to testicular deficiency
   a. Pre- and post-pubertal eunuchs
   b. Prepubertal functional (non-castrated) eunuchs
   c. Pubertal eunuchoids
   d. Postpubertal eunuchoids
   e. Male climacteric

B. *Tubular Hypogonadism*
1. Spermatogenic injury
   a. Undescended and ectopic testes
   b. Mumps orchitis
2. Hyaline tubular sclerosis
3. Adult idiopathic spermatogenic failure
4. Congenital germinal cell aplasia

C. *Impotence*

Two forms of hypogonadism may be distinguished, the total or tubulo-interstitial and the purely tubular. Intermediate degrees of hypo-activity are, quite understandably, often encountered.

A. TUBULO-INTERSTITIAL HYPOGONADISM

Depression of interstitial cell activity and androgen output gives rise to eunuchoidism with diminished 17-ketosteroids in the urine. The anatomical, physiological and psychological changes in the eunuchoid depend on the developmental stage that the individual attained before testicular function was lost.

In the presence of normal amounts of growth factor, the prepubertal eunuchoid in adult life has a characteristic tall build with excessive limb length due to late closure of epiphyses. (Span is thus greater than height, distance from pubis to soles being greater than from pubis to vertex) and digits are long and slender. Penis and scrotum maintain infantile proportions, and though scalp hair is abundant, beard, axillary hair and pubes are greatly reduced and resemble those of early puberty. In old age there is a tendency to obesity. The voice does not "break".

The post-pubertal eunuchoid may show none of the anatomical changes apart from slight penile shrinkage, and diminution of hair. Potence and libido are diminished but may persist, and obesity and gynaecomastia are common. A prematurely senile skin is often observed and is generally attributed to thyroid deficiency.

Symptomatic treatment with testosterone will eliminate the eunuchoid symptoms, but it is not necessarily the treatment of choice and in hypopituitary cases gonadotrophin treatment is more logical, and clinically more successful.

1. *Hypopituitary Hypogonadism*

Deficiency in gonadotrophin secretion produces a eunuchoid state that is associated with low levels of urinary F.S.H. and responds to treatment with I.C.S.H. Treatment with testosterone will alleviate symptoms but is to be deplored since it apparently decreases the individual's chance of being weaned from substitutional therapy.[27] Testicular biopsies in all forms of hypopituitary hypogonadism show what is essentially a prepubertal testis. Spermatogonial mitoses may be encountered, but there is no Leydig-cell differentiation, and Sertoli cells are infantile. Spermatogenesis is absent.

a. Panhypopituitary hypogonadism.

i. Juvenile panhypopituitarism. Here sexual infantilism is associated with dwarfism, myxoedema, and adrenal insufficiency.

ii. Adult panhypopituitarism, or Simmond's disease, shows similar deficiencies apart from the dwarfism. This condition is often indistinguishable from anorexia nervosa.

Treatment of panhypopituitarism at present is with thyroid, adrenal cortical extract and I.C.S.H.

b. Hypogonadotrophic hypogonadism.

i. Primary juvenile hypogonadotrophic hypogonadism. Clinically this shows a greater tendency to progeria-like wrinkling of the face, rather more abundant pubic and axillary hair and urinary 17-ketosteroids than in prepubertal non-castrated eunuchs, small testes and normal male mammae.[26]

ii. Primary post-pubertal hypogonadotrophic eunuchoidism. This has the features of a postpubertal eunuchoid, or male climacteric, with low F.S.H. output, responding to chorionic gonadotrophin.[26]

Treatment of hypogonadotrophic hypogonadism is with chorionic gonadotrophin (I.C.S.H.) 750 units intramuscularly twice daily for 6 weeks, then reduced by half and continued for 2 months. Treatment is suspended for 3 months, to see whether spontaneous improvement has commenced—as sometimes occurs; if not, then the course is repeated. Many cases of the juvenile type handled in this way maintain their improvement on this regime if it is continued for up to 18 months. Chorionic gonadotrophin leads to Leydig-cell development with Sertoli-cell maturation but no spermatogenesis; further therapy with F.S.H. has been successful in the latter regard.[26]

2. *Hypogonadism Associated with Hepatic Deficiency.*

Hypogonadism has been observed in cases with dietetic deficiencies and in cirrhosis of the liver,[33] and in cases suffering from dietetic deficiencies, e.g. in pellagra. Testicular biopsies show fibrous thickening of the membrana propria of the tubules, hyalinization and obliteration in some, and in others varying degrees of epithelial atrophy. The histological features are not those of hypogonadotrophic hypogonadism and may be attributable to excessive circulating oestrogen.[33] It is believed that the failure of the liver to inactivate oestrogen causes excessive depression of pituitary activity: an abnormally low level of F.S.H. has been found in one such case.[34]

3. *Total Hypogonadism Due Primarily to Testicular Deficiency.*

Such cases show high levels of F.S.H. in the urine. There is no response to chorionic gonadotrophin, but a definite response to androgens in adequate dosage.

a. *Pre- and post-pubertal (castrated) eunuchs.*

b. *Prepubertal functional (non-castrated) eunuchs.*

Male congenital eunuchism [35] is caused by agenesis or complete atrophy of the testes. It is sometimes familial. Both small and tall forms occur and the features are those of prepubertal castration with or without deficiency of the growth hormone. The scrotal contents are diminished, and of all eunuchoids these cases show the least development of pubic and axillary hair. Gynaecomastia is usually present, and biopsies show wolffian duct derivatives or remnants of atrophic testes.

c. *Pubertal eunuchoids.* Such patients show some signs of puberty, but development is incomplete. They represent the eunuchoidal forms of the Klinefelter syndrome of pubertal hyaline sclerosis (see below). Occasionally a eunuchoid state is encountered in men with bilateral ectopic testes.

d. *Postpubertal eunuchoids.* Bilateral tubulo-interstitial testicular damage may follow trauma, torsion, herniorraphy or infection. The prominent feature is impotence or a male climacteric, the latter developing only when previously there was normal masculinity. High F.S.H. levels are present, and biopsy shows failing Leydig-cell function.

e. *Male climacteric.* This may develop after the middle age in some men. It must be distinguished from psychoneurotic cases of impotence, where the onset of symptoms is vague, urinary F.S.H. levels are normal, and response to testosterone is absent. True cases of the male climacteric show sufficient endocrine disturbance to deserve separate classification. There is a definite time of onset of symptoms, hot flushes, and high F.S.H. levels in the urine, all changes responding within 3 weeks to testosterone therapy. The testes reveal spermatogenic reduction, with diminished size and number of Leydig-cells.[36] Normal testes with high F.S.H. levels have been reported.[37] In general the symptoms are mainly psychological, vasomotor, constitutional and sexual, and the problem of sterility seldom arises.

Substitution therapy with androgens is recommended for these cases of total primary testicular deficiency. The spermatogenic defect appears to be hopeless. The amount of androgen required will vary from case to case, but the following gives the usual dosage for the different routes of administration:

1. Intramuscular injections 3 times a week of 25–50 mg. testosterone propionate in oil.

2. Oral methyl-testosterone 25–100 mg. daily. The drug is best absorbed sublingually but is also effective when swallowed.

3. Implants of pellets of testosterone (6 tablets each of 75 mg. every 6–8 months). Pellet implantation appears to be the most convenient and economical procedure for maintenance therapy.

B. TUBULAR HYPOGONADISM

These patients show seminiferous tubular deficiency, but varying degrees of Leydig-cell damage are also encountered. Testes are usually atrophic though this is not always clinically detectable. There is no response to chorionic gonadotrophin.

The clinical features of spermatogenetic hypoplasia, whatever the aetiology, are similar. There is azoospermia or hypozoospermia, with slight, sometimes moderate eunuchoidism.[37] In the later stages urinary F.S.H. is increased in proportion to the number of tubules involved by hyalinization or lack of spermatogenesis—but this does not occur with maturation arrest.[38] This fact suggests either that the absence of spermatogenic cells modifies the function of Sertoli or Leydig cells or that the spermatogenic cells themselves secrete a hormone. Such a hormone has often been postulated and named 'androitin', 'inhibin' and 'X substance' but has been generally regarded as secreted by Sertoli cells.

1. *Spermatogenic Injury*

Trauma, infection (e.g. mumps), ectopia and X-irradiation may produce temporary (self-healing) or permanent irreversible damage. This leads to testicular conditions very similar to adult spermatogenic failure. Two of these conditions deserve special attention on endocrinological grounds.

a. Undescended and ectopic testes. Harrison [39] has drawn attention to the ingenious pre-cooling devices present in the testicular vasculature, which in man ensure that the testicular temperature is maintained about 2.2° C below that of the abdomen. It is understandable therefore that bilateral cryptorchids will be sterile; the damage affects primarily, and eventually irreversibly, the spermatogenic tissues, but also involves the Leydig cells. In some cases eunuchoidism may be quite marked. Leydig-cell hypofunction has been demonstrated in cryptorchid animals, and has been shown to respond to I.C.S.H.[40]

Rational treatment of undescended testes has been delayed because the condition is subject to a large number of variables of which the following are probably the most important:—

i. Constitutional deficiency of the retained testis. This was first suggested by John Hunter, and has often been invoked to explain failures in orchidopexy. Rea [41] has encountered unilateral anorchism in 6 cases of undescent. I have however observed permanent sterility in a case where a scrotal testis was displaced traumatically to an ectopic position, where it remained for some years, and it seems evident that prolonged exposure to an increased temperature alone is sufficient to explain most cases of permanent testicular damage of this type, as Nelson [40] has shown experimentally in rats.

ii. The second variable is thus the length of time spent in an abnormal position. Cooper [42] has claimed that she could detect histological changes in the undescended testis at 2½ years, Nelson [40] did not find them until 6. Others[41] do not agree that prepubertal undescended testes differ significantly from scrotal. My own observations support Nelson's findings.

After puberty, however, degeneration is progressive,[40, 43] spermatogonial depletion continuing until total germinal cell aplasia results [40] and even the Sertoli cell epithelium disappears.[44]

iii. Situation. The testis may lie anywhere along the normal route of descent (abdominal, canalicular or extracanalicular) or may lie elsewhere (ectopia). The testicular temperature is obviously affected by its situation, and at the external ring Cooper [42] and Rea [43] have both found that normal spermatogenesis can occur, bearing out Cooper's statement that the farther the pre-adolescent

testis has descended the more closely it corresponds to the normal gland of that age.

iv. Maldescent may or may not be associated with anatomical obstructions (bands, hernia) of varying effectiveness.

v. A tendency to spontaneous descent exists, for the incidence of cryptorchidism is 10% at birth, 2% at puberty and 0.2% in adults.[45]

vi. The susceptibility of the undescended testis to chorionic gonadotrophin also varies with age, and is maximal as puberty approaches (after 10 years).[45]

In view of the heterogenous nature of this condition and the absence of controlled long-term experiments it is not surprising that differences of opinion should exist as regards treatment, especially since some are content with a scrotal position, however atrophic the testis, and others, not unreasonably, require normal spermatogenesis as the criterion of success. Hansen[46] for example found among 25 bilaterally cryptorchid men 'successfully' treated by orchidopexy that 14 were azoospermic, 2 had counts less than 1 million/ml., 2 had counts between 1 and 10 million/ml. and 7 between 10 and 50 million/ml. That is, about two-thirds were sterile, and only 2 out of 25 showed no sign of impaired fertility.

In view of the serious and irreversible effects of post-pubertal cryptorchidism, it is clear that the testes should be in the scrotum before puberty. Bishop[45] recommends that this be achieved as soon after the age of 9 as possible, while Nelson[40] says that in view of the histological changes observed, treatment should not be postponed beyond 6 or 7.

At present it seems that chorionic gonadotrophin 500 units 3 times weekly should be tried on all cases with undescended testes unless hernia, mechanical obstruction or ectopia make surgical interference inevitable. Most succesfully treated cases respond within 3 months.[45] Where hormonal treatment fails, it seems that orchidopexy should be undertaken without delay, since Mimpriss[47] has produced experimental evidence that testicular degeneration is accelerated in a mechanically-retained testis after gonadotrophic therapy. This author reported a much greater percentage of clinically satisfactory results after the Keetley-Torek operation, as compared with Bevan or Turner procedures.

As regards orchidopexy, Harrison[39] has drawn attention to the superficial position of the testicular artery (immediately below the tunica albuginea) and the danger of including this in a ligature. Orthopaedic achievements with traction make one wonder whether more use might not be made of this manoeuvre in difficult orchidopexies.

b. *Mumps orchitis.* Bilateral mumps orchitis is rare, so that this is unlikely to be encountered as a cause of sterility. Serological tests have proved of value in diagnosis, and with their aid conclusive evidence has been found of mumps orchitis occurring without parotitis in infants.[48] The rationale of the therapeutic and prophylactic use of oestrogens in treating mumps orchitis remains to be demonstrated.[49]

The studies of Gall[50] and Charny and Meranze[51] indicate that capsulotomy is unlikely to prevent testicular damage, for the condition is associated with focal changes within the seminiferous tubules, and is not, as was one time suggested, simply a pressure atrophy due to interstitial oedema. The focal nature of the change, furthermore favours some degree of recovery.

2. *Hyaline Tubular Sclerosis* (pubertal seminiferous tubular failure).

This is the commonest hypogonadal syndrome and was described in 1942 by Klinefelter, Reifenstein and Albright.[52] Testes are usually reduced in size, and biopsy reveals hyalinization of the tubules with clumps of Leydig cells suggesting an increase in their number. F.S.H. levels are raised to the levels found in castrates. Three clinical sub-types are described by Heller and Nelson[53] depending on the degree of involvement of the Leydig cells: viz. i. The non-eunuchoid group with gross gynaecomastia, ii, the moderately eunuchoid group with slight gynaecomastia, and iii, the eunuchoid group lacking gross signs of gynaecomastia. All cases develop signs of the male climacteric by 25 years.[26] Most authors have regarded this condition as primarily tubular failure, though de la Balze *et al*,[54] have suggested that the primary disorder is in the Leydig cells, leading to fibrosis, hyalinization of tubules and secondary germinal epithelial atrophy.

The disease appears to be familial[55] and makes its appearance at puberty.

Neither the gynaecomastia nor the seminiferous tubules respond to hormones, so the treatment is plastic surgery to the breasts where necessary and testosterone pellet implants.

3. *Adult Idiopathic Spermatogenic Failure.*

Many physically normal men show a progressive depletion of their spermatogenic cells with intermediate stages of patchy spermatogenesis, diffuse spermatogenic arrest (usually at the primary spermatocyte stage) and finally complete absence of spermatogonia (spermatogenic aplasia). In many there is no tubular sclerosis or relatively little, and the Leydig and Sertoli cells appear normal. Other cases show generalized peritubular fibrosis.

Treatment of idiopathic spermatogenic failure:

i. *Testosterone rebound.* Heller *et al*[56] discovered that temporary depression of spermatogenesis by heavy doses of testosterone (25 mg. of testosterone propionate intramuscularly daily for 1–3 months) is followed 6 months later by a remarkable recovery of tubular function far beyond earlier levels. The extent of this recovery is complete 1½ years later. The precise explanation of this response is lacking and the indications for its use are also vague. In the present state of our knowledge, this method of therapy deserves trial in any case of oligozoospermia.

ii. *Nutritional and testosterone regimen.* Glass and Russell[57] have reported improvement in cases of oligozoospermia on a diet of high protein, high vitamin and liver content, with supplementary 'physiologic' doses of testosterone propionate (50 mg. intramuscularly thrice weekly for 10 doses).

iii. *Vitamin E therapy.* There is very little evidence that Vitamin E may possibly be of use[58] and much that it has proved valueless.[59, 60, 61]

4. *Congenital Germinal Cell Aplasia.*

Certain testes show unexplained complete absence of germinal cells, although Leydig and Sertoli cells are more

or less normal.[62] The F.S.H. levels in these cases are normal or slightly increased.

Comment: Hyaline and Fibrillary Tubular Membranes.

Sclerosis of the membrana propria is common to many types of atrophic tubules besides those found in Klinefelter's syndrome. Some hold that the shrinkage of the tubule follows the sclerosis, others that the sclerosis follows shrinkage. Probably both sequences may occur. Many believe that the treatment of such cases is hopeless, yet it should be remembered that in heavy dosage with testosterone hyaline membranes develop which subsequently disappear.[56] It seems that this striking change need not necessarily be irreversible, though it probably impairs epithelial nutrition very severely.

C. IMPOTENCE

Impotence used to be a common complication of diabetes mellitus but is rarer now that diabetes is better controlled. It may also develop in Addison's disease, thyrotoxicosis and myxoedema.[63] Generally, however, impotence appears as an isolated complaint and of these cases in less than 1 in 10 is it attributable to eunuchoidism. Even here psychologic causes for impotence may be present[6] since post-pubertal eunuchs are not necessarily impotent. In an impotent patient, the occurrence of masturbation, or nocturnal erections or emissions strongly suggests psychologic causes.

Testosterone is the obvious treatment for endocrinologic impotence. It is not of value in the psychologic form.

CONCLUSION

Previously patients with aplastic azoospermia or oligozoospermia could expect litle help or hope of improvement; in the last decade endocrinological investigations have demarcated definite syndromes and clarified therapeutic principles. Nevertheless it should be remembered that endocrinology offers assistance to only a proportion of sterile husbands, and there are many aspects of male infertility for which the skill of the surgeon or general practitioner is more appropriate.

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A 'SCANSORIUS' MUSCLE IN A BANTU MALE*

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During 1950, while a study of the anterior gluteal region was being carried out in the Department of Anatomy, University of the Witwatersrand, the presence of a 'Scansorius' or Gluteus Quartus muscle was observed in two cadavers, belonging to a male and a female Bantu-speaking South African Negro. The female cadaver was unfortunately due for burial before a detailed study could be made of the gluteal region; however, a distinct fourth gluteal muscle was observed in this body. In the male subject, however, it was possible to define the attachments of a similar muscle, a description of which forms the topic of this paper.

The term Gluteus Quartus is used here instead of 'Scansorius' since the former term serves to relate the muscle to the other gluteal muscles, whereas the latter term suggests a function not necessarily subserved by the muscle in man.

The arrangement and relationship of the gluteal muscles was as follows:—

Tensor Fasciae Latae (Figs. 1 and 2) possessed the usual attachments.

Gluteus Maximus (Fig. 1) was a well-developed muscle. The attachments corresponded to those described in most text-books.

Gluteus Medius (Figs. 1 and 2) was also well-developed and showed no significant variation from the usually described attachments. Near the anterior superior iliac spine, in the area from which the Tensor Fasciae Latae muscle arises, Gluteus Medius was closely related to the Tensor as well as to the Gluteus Quartus. The nerve to the Tensor passed between the Gluteus Medius and the Gluteus Quartus muscles to reach its destination. A bursa

Fig. 1. Dissection of Gluteal Region. Gluteus Maximus reflected. A—Gluteus Maximus. B—Gluteus Medius. C—Gluteus Minimus. D—Gluteus Quartus. E—Gluteal Fascia. F—Tensor Fasciae Latae. G—Ilio-Tibial Tract. H—Vastus Lateralis.

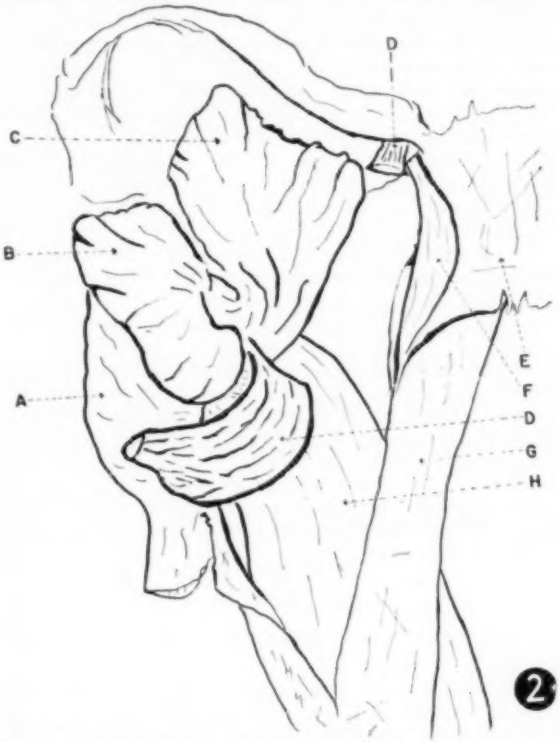

Fig. 2. Dissection of Gluteal Region. Gluteus Medius reflected. A—Gluteus Maximus. B—Gluteus Medius. C—Gluteus Minimus. D—Gluteus Quartus. E—Gluteal Fascia. F—Tensor Fasciae Latae. G—Ilio-Tibial Tract. H—Vastus Lateralis.

* A Paper read at the Annual Congress of the South African Association for the Advancement of Science held in Bulawayo in July 1953.

was observed between the Gluteus Medius and the greater trochanter.

Gluteus Minimus (Figs. 1 and 2) was fairly well developed. It was separated from the Gluteus Medius by Gluteus Quartus. Besides a few fibres ending posteriorly in the capsule of the hip-joint, Gluteus Minimus also had the usual attachments.

Gluteus Quartus ('*Scansorius*') (Figs. 1 and 2). Although a separate muscle belly, Gluteus Quartus was closely associated with the other gluteal muscles.

The origin was from the gluteal fossa in the area immediately medial to the origin of Tensor Fasciae Latae and superior to the anterior part of the middle gluteal line.

The width of the muscle belly was about one-third of that of the Gluteus Minimus. On the right side, it was closely associated with the Tensor while on the left side the proximal part was fused to the tendon of Gluteus Minimus.

The insertion was slightly behind that of Gluteus Minimus and in front of and continuous with the lower part of the insertion of Gluteus Medius. The insertion extended on to the anterior inter-trochanteric line, between the origins of Vastus Lateralis (Fig. 1) and Vastus Intermedius.

Nerve Supply. The branch of the superior gluteal nerve to the Tensor Fasciae Latae muscle, gave off a small twig to the Gluteus Quartus muscle, during its course between the latter muscle and the Gluteus Medius.

The Gluteus Quartus probably reinforced the walking or climbing functions of the Gluteus Minimus and, to a lesser extent, that of Gluteus Medius.

In addition to the study made on this male cadaver, observations were made in 27 other Bantu Negro cadavers. In most, a muscle segment corresponding to Gluteus Quartus could be partly separated. However, the muscle segment was not always a clearly defined belly, because much of it was fused with Tensor Fasciae Latae, Gluteus Medius or Gluteus Minimus.

DISCUSSION

The appearance of a fourth gluteal muscle in man is mentioned by Bryce [2] and by Thane,[3] who describe it as a variation of the Gluteus Minimus. Bryce states:

'Detached slips of the muscle may connect with the tensor fascia latae, with the superior gemellus, with the upper part of the origin of vastus lateralis or with the capsule of the hip-joint. This last may take the form of an accessory muscle under cover of the main mass. The anterior and posterior fibres may be separate or the anterior fibres detached from the rest may form an independent muscle, namely, Scansorius or Gluteus Quartus, or Invertor Femoris (Owen).'

K. von Bardeleben,[1] too, speaks of a fourth gluteal muscle—Invertor Femoris—between Gluteus Medius and Gluteus Minimus. Most standard text-books of human anatomy mention that Gluteus Minimus occasionally fuses with one or other of the related gluteal muscles.

In conclusion, it may be mentioned that comparative anatomical studies of the anterior gluteal region with particular reference to the Scansorius muscle have been made by the writer in male specimens of the chacma (Papio ursinus) and a chimpanzee, which were available in the Department of Anatomy.

The Scansorius muscle is absent in the baboon, but the anterior one-third of the gluteus medius is very thick. In the chimpanzee a well-developed Scansorius is defined.

The results of these observations together with a study of the gluteal region in Primates and a more detailed description of the observations in the other cadavers, are to be published later.

It is hoped that these studies will throw light on the probable arrangement of the gluteal muscles of the Australopithecines.

My sincere thanks are due to Professor R. A. Dart for his constant encouragement and for allowing me to carry out this work in the Department of Anatomy. I should like to add my appreciation to Dr. L. H. Wells for his guidance and helpful advice. My thanks are also due to Dr. P. V. Tobias who kindly assisted me in the final preparation of this paper. Mr. G. Blecher and Mr. A. Shevitz assisted with the illustrations and to them I am most grateful.

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USE OF ANAL SPHINCTER IN STRESS INCONTINENCE*

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After the closure of the vesico-vaginal fistula [5] following severe cases of obstetrical pressure-necrosis, the patient is often left incontinent of urine because no bladder sphincter remains.

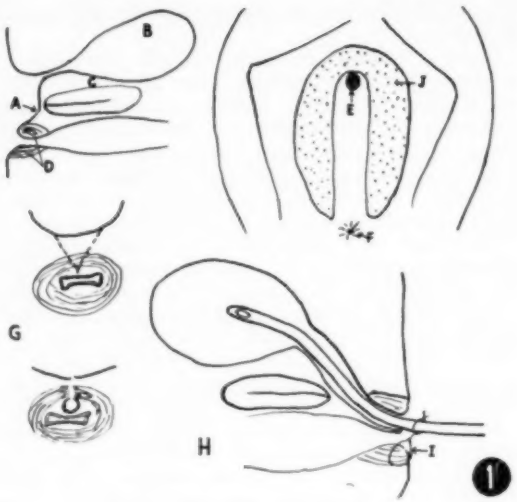
There may also be very little vagina left, with menstrual function permanently in abeyance. (Sometimes this picture presents after severe pressure-necrosis without actual fistula formation.) In such cases one has tried various forms of sling operation,[1, 2] with only occasional success, since more often than not the fistula is accidentally re-opened. In one case the Marshall Machetti operation [3] of suturing the bladder neck to the symphysis pubis was unsuccessfully tried.

In 11 cases one has closed what little remained of the vagina and brought the urethra out between anal mucosa and anal sphincter. The results as regards urinary control have been excellent (Figs. 1, 2 and 3).

* A paper read at a meeting of the Society of Obstetricians and Gynaecologists (Southern Transvaal Branch), June 1953.

Fig. 1.

(A) Remnants of vagina.
(B) Bladder.
(C) Uterus.
(D) Anal sphincter.
(E) Urethral meatus. (J) Denuded area, to be approximated around catheter.
(F) Anus.
(G) Method of incising anal sphincter and approximating it around extended urethra.
(H) Final result with catheter in position.
(I) Stitch keeping catheter in position for 14 days.



A vagina may be constructed later with the urethra and bladder below and the symphysis pubis above, using McIndoe's methods.[4]

One has tried to use this operation in severe cases of stress incontinence where there is a normal vagina, by creating a passage round the vaginal entroitus. This was not successful as the passage tended to break down, but in elderly women with postmenopausal contracture of the vagina the operation may be employed by combining it with a colpocleisis.

It is respectfully suggested that urologists could make similar use of the anal sphincter in males who have no sphincteric control of the bladder.

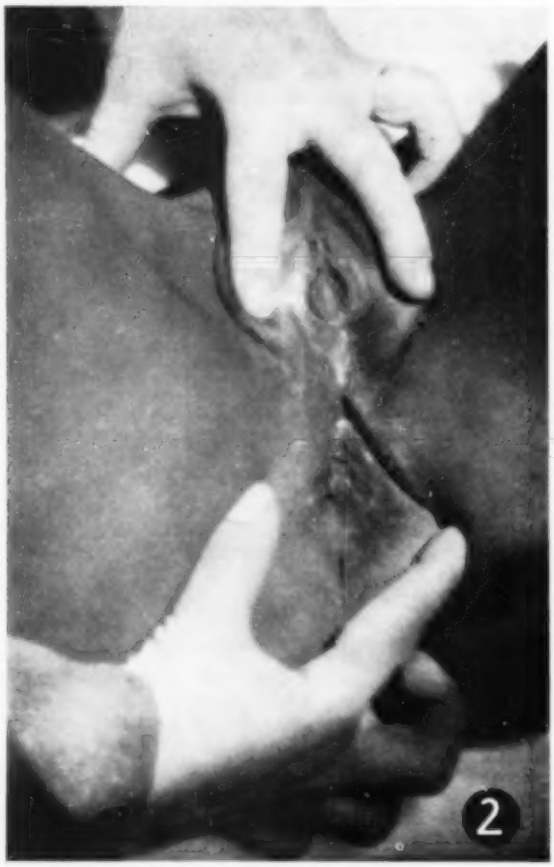


Fig. 2. Final Post-operative Appearance.

Fig. 3. Cystogram with narrow catheter forced up into bladder. Note dilated urethra as far as anal sphincter.

My thanks are due to Professor O. S. Heyns for his interest and encouragement.

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SOUTH AFRICAN PAEDIATRIC ASSOCIATION

PROCEEDINGS AT THE FIRST CONGRESS HELD IN DURBAN

The first Congress of the South African Paediatric Association was held in Durban from 20 to 22 August 1953.

The President, Dr. Seymour Heyman,n was in the Chair and the following full and associate members were present: Harvey Cohen, R. Drubin, B. Epstein, P. M. S. Fischer, Joan Griffiths, S. Kaplan Jackson, P. Klenerman, M. Medalie, B. G. Melle, B. M. Moodie, John L. Parnell, L. Sagorin, H. L. Wallace, F. Walt, C. Glynn Williams, M. Witkin. A number of delegates were accompanied by their wives.

A warm welcome was extended to two distinguished visiting Paediatricians from the United States, Dr. Harry Bakwin and his wife, Dr. Ruth Morris Bakwin, of New York. The active part which they played in the proceedings added greatly to the interest of the meetings, and it was a privilege to have them in our midst.

In opening the Congress, the President welcomed delegates and guests and read telegrams of good wishes from Johannesburg, Cape Town, Port Elizabeth and London.

The meetings were held in the Addington Children's Hospital and were very well attended. In addition to the delegates themselves, local practitioners, representatives of the Public Health and School Medical Services and senior members of the nursing profession were present at all meetings. It was gratifying to see so many of these interested in the proceedings.

PAPERS PRESENTED

The papers presented by members covered a wide field and gave rise to much lively discussion, as also did the demonstration of cases by members of the staff of the Children's Hospital.

During Congress the afternoons were left free for recreation and to enable members from the various centres to meet informally. On the first afternoon, through the courtesy of the Chief Medical Inspectors of Schools, Natal (Dr. D. B. Pauw), a number of delegates visited the Government Open Air School for physically handicapped children. The Principal, Miss Davies, conducted the visitors round the school and explained how the special educational routine was adopted to the varying limitations of the children. The visit was rounded off by a sumptuous tea which had been prepared by some of the pupils attending the class in cookery.

Visits were also made by small groups to various hospitals, child welfare and social welfare centres, and the Institute of Family and Community Health (Health Centre) at Clarewood.

Due attention was paid to the social side of the Congress. On the first evening an enjoyable braaivleis was given by Dr. and Mrs. H. L. Wallace at their residence, at which all the delegates and their ladies were present. Later the first Congress birthday cake was cut by the President amidst acclamation.

On the following evening the official Congress dinner was held at the Park View Hotel, and the Association had the honour of entertaining a number of distinguished guests.

Wives and members were entertained at a theatre party the same evening.

SCIENTIFIC COMMUNICATIONS

Dr. Harvey Cohen (Johannesburg). *Subdural Haematomata in Infants.* This paper dealt with subdural effusions in infants and a short review of the literature was given. The notes on cases of subdural haematomata were presented. The problem of subdural effusions complicating purulent meningitis was discussed and illustrative cases were reviewed. The symptomatology, diagnosis and treatment were described and a plea made for a greater awareness of the condition. It was stressed that untreated subdural effusions could cause retardation of mental and physical development.

Dr. C. Glynn Williams (Pietermaritzburg). *Rumination in a Bantu Baby.* A clinical description of ruminating in a Bantu baby, aged 5 weeks, was recorded.

As so little is known regarding the pathology of this condition, an attempt was made to demonstrate the mechanism whereby these children regurgitate their food at will. Radiological studies during several barium meals were undertaken and these showed that the primary aetiological factor is one of spasm, namely, pharyngospasm, cardiospasm and pylorospasm alone or in combination.

Gruleè has demonstrated in certain babies of the hyperkinetic type a congenital tendency towards spasm, which may manifest itself at birth or soon after, or may remain latent until some post-natal exciting factor such as dyspepsia or aerophagy is introduced. This primary spasm, once established, initiates secondary anti-peristaltic waves, which force the food up into the baby's mouth. As a result of the pleasure derived from this act it soon becomes a voluntary habit, in some lasting for a short while and of little or no significance, but in others persisting and seriously affecting the child's nutritional status.

Under the circumstances, a rational therapeutic approach seemed to be the exhibition of an antispasmodic drug; this particular child was given 'eumydrin' with a dramatic response. From more recent experiences of the author, it would appear that the combination of a sedative with the anti-spasmodic should give better results.

Dr. M. Witkin (Johannesburg). *Some Comments on Bedwetting.* The author held that enuresis should be regarded as a symptom-complex due to an irritable bladder, aggravated by emotionalism and 'parent-pecking'. The pathogenesis is mostly an achalasia of the detrusor muscle. Hypersomnia + polyuria are overrated factors.

Cure is dependent on correct psychological approach. The condition clears up at the latest, in the female, with the onset of menstruation and, in the male, before marriage.

Dr. M. Medalie (Johannesburg). *Cortisone in Nephrosis.* A case of nephrosis was described in which the response to cortisone and A C T H was most effective.

Of special interest was the relationship of a low blood albumen to a high cholinesterase, indicating an active liver function during the active phase of the disease. The proteinbound iodine was markedly depressed and an attempt was made to describe the mechanism involved.

Dr. B. G. Melle (Johannesburg). *Tuberculous Meningitis.* The history sheets, photographs and charts of 4 successfully treated cases were presented. Treatment from 1947 onwards was discussed. Streptomycin was at that time in very limited supply. Different complications occurred in each case, yet the end-results were eminently satisfactory, when reviewed several years later.

Dr. John L. Parnell (Johannesburg). *Blount's Disease (osteochondrosis deformans tibiae).* The author quoted W. P. Blount's original article (1937) in which attention was drawn to a condition occurring in infants and children causing bowing of the legs, which is non-rachitic in origin. The disease affects one or more

growth or ossification centres in children and begins as a degeneration or necrosis followed by regeneration or re-calcification.

There are two types of the disease: 1. Infantile 2. Adolescent X-ray examination is essential for the diagnosis, and the main features are: 1. Abrupt angulation just below the proximal tibial epiphysis. 2. Medially-expanded, sometimes irregular, epiphyseal line. 3. Wedge-shaped epiphysis. 4. Prominent beak-like recurving medial metaphysis. 5. Within the beak are cartilage islands.

A differential diagnosis must be made from the following:

1. Rickets. 2. Dyschondroplasia. 3. Syphilis. 4. Tuberculosis. 5. Morquio-Brailsford disease.

The X-ray films of 3 cases of this condition were shown: 1. A European male child of 4 years with the condition in its cured state. 2. A European female child of 3 years with marked osseous changes characteristic of the disease. 3. A non-European male child of 5 years with marked bowing of the legs and extensive bony changes.

Dr. B. Epstein (Pretoria). *Christmas Disease.* The author detailed the investigations and findings in a case of 'Christmas Disease', a haemophilic-like condition, yet not true haemophilia. It was due to another factor missing from the patient's blood—a factor closely related to but not identical with Factor VII.

The defect in the patient's blood was corrected by the addition of haemophilic plasma and by a normal control. The addition of plasma from another case of 'Christmas Disease' failed to correct this defect.

Infant Care

Dr. Harry Bakwin (New York). *The Psychological Aspect of Infant Care.* The many psychological problems that may arise in infant care were fully discussed. It was suggested that the newborn infant progressed better when 'roomed in' with the mother, than when left alone in the nursery. Likewise, young infants had a more rapid convalescence in hospital, if the mothers were allowed to attend them for some time daily.

While advocating breast feeding one should avoid stressing its importance to such a degree that the mother feels inadequate or guilty if she finds it impossible to nurse her baby. Success in breast feeding depends to a large extent on how strongly the mother desires to nurse and on the attitude of those around her. Apart from the psychological implications, breast-fed and bottle-fed infants show definite somatic differences in chemical constitution, metabolic response and resistance to disease.

A plea was made for a rational approach to the feeding regime. The author favoured a feeding-time schedule which, though not too rigid in its application, did not permit of completely haphazard self-demand, since the latter makes it difficult to run an orderly household. Modified self-demand feeding is recommended when relating to the amount and, later on, types of food, the intervals between feedings being kept fairly constant. Solids should be introduced when the infant's oral musculature is ready to receive these, generally at 3 to 4 months of age. Earlier feeding of solids creates mechanical difficulties on the part of the infant and the mother may then become anxious, tense and worried.

Toilet training should not be started until the child is able to sit up well and a ritual should certainly not be made of such training in the very young.

Dr. Joan Griffiths (Johannesburg). *Child Health: Johannesburg, 1950-1951.* The background of Johannesburg babies and their vital statistics were discussed and compared with those elsewhere. Infant Welfare Clinic attendance figures were given for the 4 chief racial groups. Figures were given for pregnancy order, birth weights, time of clinic attendance, incidence of prematurity, breast feeding, and incidence of some common infections in clinic babies. The effect of these factors on infant health was discussed.

Clinical Demonstration of Cases

In addition to the papers read, a demonstration of cases was given by members of the Paediatric staff of the Children's Hospital.

Dr. H. L. Wallace showed an example of the Guillain-Barrè syndrome with widespread paralysis simulating poliomyelitis, and a case of epidermolysis bullosa.

Dr. Frank Walt demonstrated the invasive phase of bilharzia (the Katayama syndrome), and 3 cases of facial palsy in children, one caused by a neuroblastoma, the second being an example of the Moebius syndrome and the third due probably to a peripheral neuritis.

Business Proceedings

The minutes of the last annual general meeting were confirmed.

The annual report and financial statement, including the Executive Committee's report, was presented by the acting Hon. Secretary-Treasurer, and was approved.

The Chairman reported that the Honorary Secretary, Dr. I. Kessel, had been on leave overseas since April and would be likely to be away for another few months. The Executive appointed Dr. John Parnell to act in his place and the Chairman expressed his appreciation to Dr. Parnell for thus stepping into the breach.

The Hon. Secretary made a statement on the forthcoming International Paediatric Congress at Havana, Cuba.

The following is a summary of some of the principal matters dealt with.

Affiliation to the International Paediatric Association. It was noted that a letter had been received from Prof. Fanconi, Secretary General of the International Paediatric Association, advising that the affiliation of the South African Paediatric Association had been confirmed.

South African National Council for Child Welfare. The Chairman reported that the constitution of the S.A.N.C.C.W. did not permit direct representation of the S.A.P.A. on the S.A.N.C.C.W., since the S.A.P.A. was a sub-group of the Medical Association of South Africa. Only national organizations could be directly represented on the S.A.N.C.C.W.; however, the M.A.S.A. made it a practice, when electing 2 members to the S.A.N.C.C.W., to request the S.A.P.A. to nominate these members for confirmation by Federal Council. In practice, therefore, the S.A.P.A. had 2 representatives on the S.A.N.C.C.W.

South African Paediatric Association Annual Prize. The Executive Committee recommended that an annual prize for final-year medical students be founded, consisting of a medal and cash award, the latter to be used for the purchase of books, instruments or subscription to medical journals. This recommendation was accepted and it was further agreed to reserve the right to withhold the award, if in any year the examiners did not consider the candidates to be of sufficient merit. Arrangements were being made to notify the Deans of the Faculties of Medicine at Cape Town, Witwatersrand and Pretoria, the subject for this year being, *A discussion of the pathogenesis, signs and symptoms, treatment and prognosis of tuberculous meningitis in childhood.*

Change in Constitution. In response to a circular letter received from the Secretary of the M.A.S.A., it was agreed to incorporate the following paragraph in Clause 5 of the Constitution:

> '*Honorary Members*—Persons of eminence who are not domiciled in the Union, provided they are medical practitioners and members of their national medical associations, may be appointed by a majority of votes of the Executive committee as honorary members.'

The text of this addendum was to be forwarded to the M.A.S.A. and would become effective after being confirmed by Federal Council.

Prize for Nurses. It was agreed that a token prize of a book be awarded to the candidate obtaining the highest marks in the annual examination for the Certificate of Paediatric Nursing, conducted by the South African Nursing Council, the course for which was at present being conducted at the Transvaal Memorial Hospital for Children.

World Conference on Medical Education. The Hon. Secretary reported that Dr. W. Rabkin had been appointed by the Executive to represent the Paediatric Association at the World Conference on Medical Education. Dr. Rabkin would be requested to submit a report to the Paediatric Association.

Election of New Members. The following were elected to membership of the Association: Full members—Dr. L. Pannall (Pretoria), Dr. L. Sagorin (Johannesburg), Dr. S. Wayburne (Johannesburg), Dr. C. Glynn Williams (Pietermaritzburg). Associate members—Dr. Harvey Cohen, Dr. J. Griffiths, Dr. I. B. Kreher, Dr. P. E. du Toit, all of Johannesburg.

Election of Office Bearers. The following were elected for the year 1953-54: President, Dr. Seymour Heymann: Hon. Secretary-Treasurer, Dr. I. Kessel. Executive Committee: Dr. B. Epstein, Dr. P. M. S. Fischer, Dr. B. G. Melle, Dr. I. Mirvish (co-opted), Dr. J. W. Rabkin, Dr. H. L. Wallace.

Matters arising from Congress. It was agreed to request the Editor of the *South African Medical Journal* to consider publishing in the *Journal* the Proceedings of Congress, including a brief

note on the social aspects, in addition to the scientific and business transactions. Further, it was decided to sound the Editor on the feasibility of publishing a special Paediatric issue of the *Journal*, this to include those papers read at Congress which he deemed suitable for such publication.

Next Meeting. It was agreed to hold a similar Paediatric Congress every 2 years, so timed as not to occur in the year in which the Medical Association held its general Congress. Selection of the venue and date for the next Congress was left to the discretion of the Executive Committee.

The President thanked all concerned in the organization of this first Congress, as also all those who had attended and thus contributed materially to the success which Congress had obviously proven to be. He particularly thanked the visitors from overseas and hoped they would in the no distant future rejoin their South African colleagues at a similar Congress.

IN MEMORIAM

Dr. C. J. Watson

Cecil John Watson was born in Cape Town in November 1905. His father, John Watson, who came of a Northumberland family and settled in Cape Town early in the present century, was a sea-captain and became managing director of the Cape Town Stevadoring Company.

Dr. Watson was educated at Diocesan College, Rondebosch, and Caius College, Cambridge. He qualified in 1932 at St. George's Hospital, London, where he held appointments as house physician and house surgeon.

He returned to Cape Town in 1933 and started practice in the Gardens in partnership with the late Dr. Frank Morris. He was medical officer to several insurance companies and to the Ladies' Christian Home, Vriede Street. He was appointed to attend the families of naval personnel. Dr. Watson was specially interested in psychology.

In the Second Great War Dr. Watson served in the South African Medical Corps and was attached to the South African Air Force. He served through the Abyssinian Campaign, and was at the landing at Madagascar, where he was visiting H.M.S. Ramillies when she was torpedoed. He was sent to Egypt to work on air-pilots' 'black-out'. After being in medical charge of various air-stations in the Union, in the last months of the war, wanting more foreign service, he joined the South African Naval Service and was appointed to H.M.S.A.S. Swale. He was twice mentioned in despatches.

Dr. Watson was well known in the Civil Service Club, on the Committee of which he served for some time. His chief interests were reading and conversation. He delighted in children and loved all forms of beauty. His many friends were familiar with his keen sense of humour.

Dr. Watson died suddenly on 5 December 1953.

RESIDENTS AT LONDON HOUSE

The Warden of London House, Guilford Street, W.C.1, Mr. E. C. Pepper, announces that there are now 56 South Africans in residence. Since the start of London House in 1930 there have been 666.

The following are among successes gained recently by South African residents:

Membership of the Royal College of Physicians (Lond.): 2.
Membership of the Royal College of Physicians (Edin.): 2.
Fellowship of the Royal College of Surgeons (Eng.) Primary: 1
Fellowship of the Royal College of Surgeons (Edin.): Primary: 1
Membership of the Royal College of Obstetricians & Gynaecology: 3.
Diploma in Child Health (D.C.H.): 4.
Diploma in Clinical Pathology (D.C.P.): 1.
Diploma in Laryngology and Otology (D.L.O.): 1.
Diploma Medical Radio-Diagnosis (D.M.R.D.): 2.
Diploma in Public Health (D.P.H.): 1.

Many ex-residents and others interested in London House were in London for the Coronation.

PASSING EVENTS : IN DIE VERBYGAAN

Brigadier Albert Sachs, C.B.E., M.D., M.Sc., M.R.C.P., Honorary Physician to the Queen, has vacated the appointment of Director of Pathology at the War Office and has been appointed Deputy Director of Medical Services of a command in the United Kingdom with promotion to the rank of Major General. Genera Sachs, who is a brother of Dr. S. B. Sachs of the Evaton Health Centre, was born in Pretoria and was educated at the Witwatersrand University before proceeding to Trinity College, Dublin.

Professor L. J. te Groen, Professor van Ginekologie en Obstetrie, Universiteit Pretoria, is tot *Fellow of the Royal College of Obstetricians and Gynaecologists* (*F.R.C.O.G.*) verkies.

Elected to Medical Council

In the list of those recently elected to the Medical and Dental Council the name of Dr. Maurice Shapiro was by error omitted Dr. Shapiro was one of the ten elected to represent the medical practitioners.

BOOK REVIEWS : BOEKRESENSIES

Clinical Allergy

A Manual of Clinical Allergy. By John M. Sheldon, M.D., Robert G. Lovell, M.D. and Kenneth P. Mathews, M.D. (Pp. 413 + xv, with illustrations. South African price: £3 12s. 3d.) Philadelphia and London: W. B. Saunders Company. South African representatives: P. B. Mayer, Cape Town. 1953.

Contents: 1. Immunochemical Aspects of Allergy. 2. Medical Evaluation of the Allergy Patient. 3. Skin Testing and Additional Laboratory Procedures. 4. Hay Fever, Allergic Rhinitis and Bronchial Asthma. 5. Procedure for Hyposensitization. 6. Constitutional Reactions, Serum Sickness and Status Asthmaticus. 7. Aerosol Therapy, Bronchoscopy and Bronchography. 8. Useful Drugs in Allergic Diseases. 9. Drug Allergy. 10. Food Allergy and Gastrointestinal Allergy. 11. Dermatologic Aspects of Allergy Practice. 12. Patch Testing. 13. Evaluation of Headache in Allergy Practice. 14. Ophthalmic Allergy. 15. Aeroallergens I: Pollen Identification. 16. Aeroallergens II: Fungus and Mold Identification. 17. House Dust and Miscellaneous Inhalant Allergens. 18. Endocrine Aspects of Allergy. 19. Vascular Allergy and Collagen Disease. 20. Psychologic Aspects of Allergy. Appendices. Index.

The place of allergy in medicine is still unsettled. Few medical men can fail to have noticed the great increase in 'allergic' complaints. With this increase has come, inevitably, a spate of books on allergy.

The volume under review is primarily concerned with the practical and technical aspects of allergy. The authors point out that there are already many text-books covering the theories, symptomatology and diagnosis of allergic diseases, and they deliberately content themselves with brief outlines in this respect. Nevertheless the practitioner faced with an emergency such as status asthmaticus will find extremely clear and thoroughly sound directions for dealing with the situation.

The chapters devoted to the carrying out of skin tests and their interpretation are particularly well handled. Very full directions are given, and probably few doctors, unless exclusively engaged in this speciality, will want to read the minute directions for the preparation of protein extracts. It is a little disturbing to find it stated that the doctor who bothers to prepare his own extracts will find it to be a profitable side to his allergic practice.

The book abounds with full and beautifully arranged tables which make the tracking down of contactants in dermatological allergy a less haphazard business than it often becomes. Equally useful are the suggestions for handling suspected food-allergy.

One of the best and most useful chapters is devoted to drugs which may be of use in treating allergic patients. The grouping of the anti-histamitics is an excellent idea and makes this difficult pharmacological subject much easier to understand. Naturally the references to plant and pollen sensitivity are not as useful to workers outside the U.S.A., and the same observation applies to many of the excellent illustrations.

The book conforms to the high standard we have come to associate with the best medical texts coming from the States. The style is unambiguous and makes for easy reading and the tabulation for easy reference.

The fact that the book will probably only appeal to a small number of readers in this country does not detract one whit from it's usefulness.

N.V.S.

ITALIAN REVIEW OF TUBERCULOSIS

Rivista della Tubercolosi e delle Malattie dell'Apparato Respiratorio. Edited by C. Cattaneo, G. Fegiz and L. Pigorini. First issue of a new Italian Journal, under the direction of Attilio Omodei Zorini. (Pp. 1-132 with illustrations. Annual Subscription $8.00) Rome: Edizioni Mediche e Scientifiche. 1953.

Contents: (by different contributors). 1. Problemi attuali della terapia biologica della tubercolosi. 2. Semeiologia e clinica della tubercolosi bronchiale. 3. Ricerche sulla resistenza crociata. 4. Le possibilita della tecnica stratigrafica nello studio delle stenosi bronchiali neoplastiche e non neoplastiche. 5. Moderni orientamenti sull'assistenza post-sanatoriale. 6. Emosiderosi polmonare (editoriale).

The medical library finds that at present new medical journals make their appearance at the rate of one per week. This is a frightful state of affairs and should restrain people from starting new adventures unless they serve a most definite and useful purpose.

That is hardly the case with this new Italian review of tuberculosis and diseases of the respiratory system. The object of the journal is stated to be the publication of original work from the tuberculosis clinic of the University of Rome and the Carlo Forlanini Institute. There are already a number of journals in the Italian language in which such works could suitably be published, such as *Annali dell'Instituto Carlo Forlanini, Lotta contro la tubercolosi, Archivia di tisiologia, Rivista di patologia e clinica della tubercolosi* etc. It will be noticed that the Forlanini Institute has now 2 journals, which is really too much.

The 1st article is a long preamble by the editor on the biological treatment of tuberculosis; it is the reprint of an official lecture given at the University of Rome and has all the sonorous qualities of such a speech. Next comes a description of bronchial tuberculosis which the author quite convincingly maintains is an important complication of collapse therapy. The 3rd article describes an apparently fine piece of work (this reviewer has no particular knowledge on this subject) demonstrating the non-development of cross-resistance of different strains of Koch's bacilli to a number of therapeutic agents. Then follows another reprint of a lecture on the comparison of tomography and other methods of X-ray examination in bronchial lesions. The lecture is described as short, but it takes up 16 pages. The 5th article is again of a general nature on post-sanatorial assistance to tuberculosis patients with special reference to legislation in Italy; and lastly there is an editorial on pulmonary haemosiderosis.

Each article is followed by a summary in different languages, so short and so full of spelling mistakes that it is virtually valueless; but as the journal must surely be meant for internal consumption in Italy—apart from constituting an edifice for the Forlanini Institute, as stated in the foreword—it does not really matter.

The journal is well printed on glossy paper and the illustrations are perfect. It has the same irritating scatter of advertisements in the middle of the text as our own *Journal* presumably necessary for economic reasons.

There are vague rumours of an international editing board for medical writing; will it ever be able to stay the avalanche?

O.E.B.—O.

CORRESPONDENCE : BRIEWERUBRIEK

PREGNANCY WITH THE PITUITARY-ADRENAL SYNDROME

To the Editor:

I have recently seen two women with the Pituitary-Adrenal Syndrome viz adiposity of the pituitary type, hirsuties with male distribution of the hair, and relative infertility.

They were married for 11 and 8 years respectively before they became pregnant. Both had negative frog tests for pregnancy (which were repeated after 14 days and again found negative), and their pregnancies terminated normally.

I should be glad to know from readers whether such an association has been noted before.

Dr. J. Helman.

4 Joubert Street,
Off Voortrekker Road,
Vasco,
Cape Town,
14 December 1953

FUNCTIONAL RELATIONTHIPS IN CLINICAL MEDICINE

To the Editor:

I have read with great interest Dr. C. F. Krige's article: *The Problem of Lower Abdominal Pain in Women*, which appeared in the *Journal* of 26 September 1953. Dr. Krige's broad and comprehensive approach, and his appreciation of the interdependence of the factors contributory to this problem will surely not fail to attract the attention of philosophic minds both in this country and beyond. For some years now, by grace of Professor E. H. Cluver, Head of the Department of Preventive and Social Medicine of the University of Witwatersrand, I have been projecting, in lectures to undergraduate and post-graduate students, the concept of functional relationships in the field of clinical medicine. Dr. Krige's paper is a good example of how clinical data should be assembled, analysed, and integrated, and for that reason it may be utilized with advantage to illustrate the newer philosophic concepts which are dramatically impinging upon the contemporary medical world.

L. F. Freed.

2 Barbican Buildings,
Opposite City Hall,
Johannesburg,
14 December 1953.

MEDICAL COUNCIL ELECTION

To the Editor:

Allow me through the correspondence columns of the *Journa* to thank most sincerely all the numerous colleagues who voted for me in the recent South African Medical and Dental Council elections.

Membership of the Council is by no means a sinecure and carries with it great responsibility. Its members have many unpleasant, unenviable tasks to perform and have to make very important decisions.

It must be remembered that the Council is a Government statutory body established to carry out the provisions of the Medical, Dental and Pharmacy Act, which was enacted by Parliament for the benefit of the public. Members of the Council must act in a more or less dual capacity. They must, as members of the public, watch after the interests of the public, while those who are also members of the medical and dental professions must naturally be interested in the welfare of these professions.

I am one of those who believe that the interests of both are complementary and not conflicting. I am of opinion that for satisfactory medical and dental services the public must be given a fair deal by the professions. On the other hand the public must realize that a satisfactory service will be possible only if these professions are also given a fair deal. Satisfied contented professions are essential for good services.

Members of the Council therefore are called upon to 'play the game' in trying to give both sides a fair deal. That at any rate is the principle on which I have tried to act during my 5 years' term of office. The fact that the members of the medical profession have again returned me to the Council encourages me to think that my past activities have been approved. The fact that I received so many votes has acted also as a helpful stimulus and as an encouragement to try and carry on with the task which my colleagues evidently wish me to undertake.

To my colleagues I say again, 'Thank you'.

James Black.

89 Lister Buildings,
Jeppe Street,
Johannesburg,
14 December 1953.

The Medical Association of South Africa
Die Mediese Vereniging van Suid-Afrika

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(1549) Specialist about to commence practice desires to share rooms with colleague in centre of town.

* * *

DURBAN

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(PD23) Natal. Prescribing practice particularly suitable for a woman doctor interested in obstetrics and gynaecology. Total gross receipts for 1950, £1,570; 1951, £1,595; 1952 (6 months), £1,340; 1953 (3 months), £382. Premium £1,250, includes furniture, fittings, instruments, drugs and existing book debts.

(PD24) Natal South Coast. Practice suitable for doctor who does not want full-time work. £250 for drugs, dressings, instruments, etc. No charge for goodwill. Small house on ½ morgen, £1,600. Immediate occupation.

LOCUMS REQUIRED

(72) Durban. Locum required for January and February with view to assistantship. General practice. Salary to be discussed with the Principal.

(73) Near Durban. Locum for January and February. £2 12s. 6d. per day, all found. Must have own car.

(74) Zululand. Locum for February. £2 12s. 6d. per day, all found. Own car necessary.

(75) Durban. 1 January. Locum view toassistantship/partnership. General practice. Salary to be discussed.

JOHANNESBURG

Medical House, 5 Esselen Street, Telephone 44-9134-5, 44-0817
Mediese Huis, Esselenstraat 5, Telefone 44-9134-5, 44-0817

INSTRUMENTS FOR SALE

(1/055) ZEISS Comberg slit lamp corneal microscope. Complete as new. Price £130.

(1/056) Bausch & Lomb microscope. Condition as new. What offers?

(1/058) (i) A complete set of P.M. Instruments in Mahogany box (Arnold & Sons). Never been used. Price £17 10s. o.n.o. (ii) Leitz Cystoscope. Hardly ever been used. One channel for catheter only. Complete with catheters in very good working order. Price £20. (iii) Portable Minnitt. Gas-Air anaesthetic apparatus as used in midwifery, with extra bag attachment. Good as new. Price £25.

ASSISTENTE-PLAASVERVANGERS VERLANG
ASSISTANTS/LOCUMS REQUIRED

(L/V491) Assistent om op 1 Februarie te begin. Verkieslik iemand met ondervinding. Salaris £120 p.m. Moet eie losies en petrol betaal. Laasgenoemde ongeveer £10 p.m. Moontlikhede van vennootskap vir regte persoon. Randse hospitaaldorp.

(L/V498) Naby Johannesburg. Assistent om so spoedig moontlik te begin. Chirurgie word gedoen. Verkieslik iemand met ondervinding. Salaris, toelaes en kommissie om gereël te word. Vooruitsigte van vennootskap.

(L/V480) Reef hospital town. Assistant to start as soon as possible. Preferably experienced man. Anaesthetic experience will be a recommendation.

(L/V486) O.V.S. Plaasvervanger vanaf 26 Mei tot 1 Augustus. Salaris £2 10s. 0d. per dag, vry losies, petrol en olie *of* 'n kar kan verskaf word. Getroude persoon kan in hoof se huis woon. Sal ook 'n dame pas.

(L/V489) Reef town. Locum for February. Terms £3 10s. 0d. per day. Mainly day practice. Locum could live in Johannesburg.

(L/V503) Reef, native practice. Locum for three weeks as from 23rd January. No night work. Own car necessary. Locum could live in Johannesburg.

PRAKTYKE TE KOOP/PRACTICES FOR SALE

(Pr/S98) Ou Vrystaatse praktyk. Verpleeginrigting waar heelwat chirurgie onderneem kan word. Een aanstelling. Die *netto* jaarlikse inkomste oorskry £2,300. Woonhuis te huur teen £12 p.m. Premie verlang is £1,300 en sluit medisyne ter waarde van £500, instrumente £200 en meubels £120, in. Terme kan gereël word.

(Pr/S99) North of Johannesburg. Well-established practice. Large European branch, also Native cash branch. Gross income £6,500 p.a. Will suit two doctors. Premium for outright sale £4,750 and includes furniture and instruments and X-ray and screening units. Long introduction (approximately 6 months) will be given. Partnership with view to taking over the whole practice will be considered.

REQUIRED

Partnership or locum or assistantship, both with view to partnership, required in Johannesburg, by doctor with own car, and a surgery in the Northern Suburbs.

Industrial Health Adviser

FRIEND NEWSPAPERS LIMITED

Applications are invited from medical practitioners resident in Bloemfontein for the part-time post of Industrial Health Adviser to The Friend Newspapers Limited. Duties would include periodic examination of employees as a matter of preventive medicine, instruction in hygiene, first-aid, home-nursing, dietetics, domestic science and prevention of occupational disease, for a monthly fee of £27 10s. 0d. In addition occasional visits to members of families of Non-European employees at a cost of 10s. per visit. The Company will bear the cost of all medical drugs and requisites.

Apply in writing to:

The General Manager,
The Friend Newspapers Limited,
P.O. Box 245,
Bloemfontein.

(This appointment has the approval of the Medical Association of South Africa—Associate Secretary M.A.S.S.)

Bethlehem Municipality

VACANCY: PART-TIME MEDICAL OFFICER OF HEALTH

Applications, endorsed "Part-time M.O.H." are hereby invited from competent bilingual and duly qualified medical practitioners to fill the abovementioned vacancy.

The salary attached to the post is £720 including cost-of-living allowance, per annum, plus £60 per annum transport allowance.

The conditions of service and appointment attached to the post will be as detailed in Circular No. 18 of 1949 of the Union Health Department, a copy of which can be obtained from the undersigned.

Full particulars regarding qualifications and experience must be submitted on the Council's official application form (obtainable from this office) and must be lodged with the Town Clerk, Bethlehem, not later than 12 noon on Monday 1 March 1954. Canvassing for appointment, directly or indirectly will disqualify a candidate.

O. S. WARREN,
Town Clerk.

Bethlehem,
11 December 1953.

Siekefonds van die Suid-Afrikaanse Spoorweë en Hawens

AANSTELLING VAN SPOORWEGDOKTER: BUTTERWORTH

Aansoeke word van geregistreerde mediese praktisyns ingewag vir aanstelling in die betrekking van spoorwegdokter, Butterworth, en die trajek Eagle (uitsluitend) tot by Mpuluse (uitsluitend), teen 'n salaris van £183 per jaar, plus die gelde en toelaes wat in die Regulasies van die Siekefonds voorgeskryf word, en met die reg om privaat te praktiseer.

Die salaris is onderhewig aan wysiging in ooreenstemming met die sensus van lede wat op 1 April van elke jaar afgeneem moet word.

Die aanstelling geskied kragtens die regulasies van die Siekefonds, en opsegging van dienste is onderworpe aan vier maande kennisgewing deur een van beide partye.

Die suksesvolle applikant moet te Butterworth woon, diens aanvaar op 'n datum wat gereël sal word, en sy pligte ooreenkomstig die regulasies van die Siekefonds uitvoer.

Aansoeke moet die Distriksekretaris, Oos-Kaaplandse Distriksiekefondsraad, Terminusstraat 19, Oos-Londen, nie later nie as 30 Januarie 1954 bereik en applikante moet die volgende vermeld:

1. Volle naam.
2. Kwalifikasies (waar en wanneer verkry).
3. Ondervinding (waar en wanneer verkry en opgedoen).
4. Datum van geboorte.
5. Land van geboorte.
6. Getroud of ongetroud.
7. Of ten volle tweetalig.
8. Of Suid-Afrikaanse burger.
9. Watter staatsbetrekking, indien enige, beklee word.

Werwing deur of ten behoewe van enige applikant stel so 'n applikant bloot aan diskwalifikasie.

Enige verder besonderhede wat verlang word kan op aanvraag van die Distriksekretaris by die bovermelde adres verkry word.

P. J. Klem
Hoofsekretaris

Johannesburg
2 Januarie 1954

Conradie Hospital, Pinelands

VACANCY: HONORARY ANAESTHETIST

Applications are invited from registered medical practitioners under the age of 60 years for appointment to the abovestated post.

The appointment will be for a period of 3 years with effect from 1.2.1954. In all other respects the appointment is subject to the Hospitals Ordinance No. 18 of 1946 (Cape), as amended, and to the regulations framed thereunder.

Applications, stating full particulars of age, qualifications, experience etc., must be addressed to the Medical Superintendent, Conradie Hospital, Pinelands, to reach his office not later than 16 January 1954.

Bethlehemse Stadsraad

VAKATURE: DEELTYDSE STADSGENEESHEER

Aansoeke, gemerk „Deeltydse'Stadsgeneesheer" word ingewag van bevoegde tweetalige en behoorlik gekwalifiseerde mediese praktisyns om aanstelling in bogenoemde betrekking.

Die salaris verbonde aan die pos is £720 per jaar, lewenskostetoelaag ingesluit, plus 'n vervoertoelaag van £60 per jaar.

Die diens- en aanstellingsvoorwaardes verbonde aan die pos is soos uiteengesit in omsendbrief Nr. 18 van 1949 van die Unie Departement van Gesondheid en 'n afskrif hiervan kan van ondergetekende verkry word.

Volle besonderhede aangaande kwalifikasies en ondervinding moet verstrek word op die amptelike aansoekvorm van die raad (verkrygbaar van hierdie kantoor) en moet die stadsklerk, Bethlehem, bereik voor 12 uur middag op Maandag, 1 Maart 1954. Stemwerwing, hetsy direk of indirek, sal 'n kandidaat vir aanstelling uitskakel.

O. S. WARREN,
Stadsklerk.

Bethlehem,
11 Desember 1953.

Public Service Vacancies

1. The attention of Medical Practitioners and Dentists registered with the South African Medical and Dental Council is drawn to an advertisement appearing in the Government Gazettes of 18, 24 and 31 December 1953, inviting applications for the undermentioned posts.

Post	*Salary Scale*	*Department*
District Surgeon Grade III (Bronkhorstspruit).	£1,020 × 60—1,380	Health. (Closing date 23 January 1954).
Dentist (Merebank, Natal).	£1,020 × 60—1,200	Health. (Closing date 29 January 1954).

2. In addition to salary a cost of living allowance at the rate of £234 per annum is at present payable to married officers.

3. It is emphasised that full particulars of qualifications and previous experience must be furnished but original certificates and testimonials should not be submitted. Application forms Z. 83 and P.S.C. 8 (*a*) are obtainable from the Secretary for Health, Pretoria, to whom filled in forms must be addressed.

(43730)

Conradie Hospitaal, Pinelands

VAKATURE: ERE-NARKOTISEUR

Aansoeke word ingewag van geregistreerde mediese praktisyns onder die ouderdom van 60 jaar, om aanstelling in bogenoemde betrekking.

Die aanstelling sal vir 'n tydperk van 3 jaar met ingang van 1 Februarie 1954 geldig wees. In all ander opsigte geskied die aanstelling kragtens die Kaapse Ordonnansie op Hospitale No. 18 van 1946, soos gewysig, en die regulasies daarvolgens opgestel.

Aansoeke waarin volle besonderhede insake ouderdom, kwalifikasies, ondervinding ens. gemeld word, moet gerig word aan die Mediese Superintendent, Conradie Hospitaal, Pinelands, en moet sy kantoor bereik nie later nie as 16 Januarie 1954.

Provinsiale Administrasie van die Kaap die Goeie Hoop

HOSPITAALDEPARTEMENT

HOSPITAALRAADSDIENS: VAKATURE

1. Aansoeke word ingewag van geregistreerde geneeshere vir aanstelling tot die volgende vakante pos:

Inrigting	*Pos*	*Emolumente*	*Sluitingsdatum*	*Aansoeke moet gerig word aan*
Somerset-hospitaal, Groenpunt	Geneesheer, Graad C. (Mediese Superintendent)	£1,000x50–1,200 p.j.	21.1.54	Die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad.

2. Die diensvoorwaardes word voorgeskryf ingevolge die Ordonnansie op Hospitaalraadsdiens nr. 19 van 1941, soos gewysig, en die regulasies wat daarkragtens opgestel is.

3. Benewens die salarisskaal soos aangedui is 'n lewenskostetoelae betaalbaar aan voltydse beamptes en werknemers teen bedrae wat van tyd tot tyd deur die Administrateur vasgestel word.

4. Van die geslaagde kandidate sal dit vereis word om 'n ongemeubileerde huis of kwartiere wat by die hospitaal verskaf word gratis te bewoon, of as 'n huis of kwartiere nie beskikbaar is nie, 'n huis te bewoon wat deur die Departement goedgekeur is ten opsigte waarvan die Departement 'n bedrag van hoogstens £180 per jaar tot die huur sal bydra.

5. Die geslaagde kandidaat, indien nie reeds in die Hospitaalraadsdiens nie, moet bevredigende geboorte- en gesondheidssertifikate indien.

6. Aansoek moet gedoen word op die voorgeskrewe vorm (Staf 23) wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, of by die Mediese Superintendent van enige provinsiale hospitaal of by die Sekretaris van enige Skoolraad in die Kaapprovinsie.

7. Applikante moet die vroegste datum meld waarop hulle diens kan aanvaar.

A562862

City of Cape Town

VACANCY FOR RESIDENT MEDICAL OFFICER

CITY HOSPITAL FOR INFECTIOUS DISEASES

Applications are invited from registered medical practitioners under 45 years of age for the above position at a commencing salary of £900 per annum on the salary scale £900+50—£1150, less £226 per annum for quarters, rations, light, fuel and laundry, plus temporary non-pensionable cost of living allowance.

Experience in modern methods of treatment of infectious diseases and tuberculosis will be a recommendation.

The successful applicant will be required to devote the whole of his/her time to the service of the Council and the appointment will be subject to the provisions of Municipal Ordinance No. 19 of 1951, the Standing Orders and regulations of the Council and the conditions of service as laid down in the Municipal Staff Code, all as amended from time to time.

Applications must be made in duplicate on the prescribed forms obtainable from the Senior Staff Officer, 2nd Floor, Municipal Buildings, Longmarket Street, Cape Town, and should reach him not later than 15 January 1954.

Canvassing of Councillors will be a disqualification.

M. B. WILLIAMS
Town Clerk
City Hall, Cape Town

21 December 1953

(17533)

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT

HOSPITAL BOARD SERVICE: VACANCY

1. Applications are invited from registered medical practitioners for appointment to the following vacant post:

Institution	*Post*	*Emoluments*	*Closing Date*	*Applications must be addressed to*
Somerset Hospital, Green Point	Medical Practitioner, Grade C (Medical Superintendent)	£1,000x50–1,200 p.a.	21.1.54	The Director of Hospital Services, P.O. Box 2060, Cape Town.

2. Conditions of service are prescribed in terms of Hospital Board Service Ordinance No. 19 of 1941, as amended, and the regulations framed thereunder.

3. In addition to the scale of salary indicated a cost of living allowance at rates prescribed from time to time by the Administrator is payable to whole-time officials and employees.

4. The successful candidate will be required to occupy, free of charge, an unfurnished house or quarters provided at the institution or alternatively, if a house or quarters are not available to occupy a house approved by the Department in respect of which the Department will contribute an amount of not exceeding £180 per annum towards the rental.

5. The successful candidate, if not already in the Hospital Board Service, will be required to submit satisfactory birth and health certificates.

6. Application must be made on the prescribed form (Staff 23), which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.

7. Candidates must state the earliest date on which they can assume duty.

A562862)

Provincial Administration of the Cape of Good Hope

UNIVERSITY OF CAPE TOWN: JOINT MEDICAL STAFF FOR GROOTE SCHUUR AND OTHER TEACHING HOSPITALS

VACANCY

1. Applications are invited from registered medical practitioners (registered specialists) for appointment to the following vacant post:

Medical Practitioner, Grade D: Department of Neuro-Surgery with salary on the scale £1,200x50–1,500 p.a.

2. The conditions of service are prescribed in terms of Hospital Board Service Ordinance No. 19 of 1941, as amended and the regulations framed thereunder.

3. In addition to the scale of salary indicated cost-of-living allowance at rates prescribed from time to time by the Administrator is payable to whole-time officials and employees. The present rate is £320 per annum for married persons and £100 per annum for single persons.

4. (*a*) The Joint Medical Staff is required to serve jointly the Provincial Administration of the Cape of Good Hope and the University of Cape Town.

(*b*) A session shall be four hours per week, not necessarily continuous, of clinical and or teaching work.

5. (*a*) Candidates must state whether they wish to be considered for—

(i) appointment in a full-time capacity; or

(ii) appointment in a part-time capacity.

(*b*) Should they wish to be considered for appointment in a part-time capacity, the maximum number of sessions which they would on appointment be prepared to undertake should be stated.

6. Candidates must be registered as a specialist in the specialty in which the vacancy exists.

7. The successful candidate if not already in the Hospital Board Service will be required to submit satisfactory birth and health certificates.

8. Application should be made on the prescribed form Staff 23 which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.

9. The completed application forms should be addressed to the Director of Hospital Services, P.O. Box 2060, Cape Town, and must reach him not later than 22 January 1954. Candidates must state the earliest date on which they can assume duty.

(A562863)

Nasionale Hospitaal Bloemfontein

VAKATURE

Aansoeke word hiermee ingewag van kandidate met geskikte kwalifikasies vir die volgende pos by die Nasionale Hospitaal en Tempe Provinsiale Hospitaal, Bloemfontein.

Aansoeke moet gerig word om die Geneesheer-Direkteur op of voor 20 Januarie 1954 te bereik en moet volle besonderhede bevat aangaande die ouderdom, professionele kwalifikasies, ondervinding en huweliksstaat van die applikant en moet voorts 'n aanduiding bevat van die vroegste datum waarop dienste aanvaar kan word indien aangestel.

Registrateur in Narkose met salaris van £400 na £600 per jaar na gelang van vorige ondervinding, plus vry inwoning op Hospitaal-terrein. Applikante vir hierdie pos moet minstens twee jaar gekwalifiseerd wees.

Van die persone wat aangestel word, sal verwag word om bevredigende sertifikate in te dien aangaande kwalifikasies.

Benewens jaarlikse salarisse ontvang werknemers op die oomblik lewenskostetoelae.

Die aanstelling geskied in terme van die Hospitaalregulasies soos gewysig.

J. W. Wessels
Geneesheer-Direkteur

15 Desember 1953

(108111)

Proviniale Administrasie van die Kaap die Goeie Hoop

UNIVERSITEIT VAN KAAPSTAD: GESAMENTLIKE MEDIESE PERSONEEL VIR GROOTESCHUUR EN ANDER OPLEIDINGSHOSPITALE

VAKATURE

1. Aansoeke word ingewag van geregistreerde geneeshere (Geregistreerde Spesialiste) vir aanstelling tot die volgende vakante pos:

Geneesheer, Graad D: Departement van Neuro-chirurgie met salaris volgens die skaal £1,200x50–1,500 per jaar.

2. Die diensvoorwaardes word voorgeskryf ingevolge die Ordonnansie op Hospitaalraadsdiens nr. 19 van 1941, soos gewysig, en die regulasies wat daarkragtens opgestel is.

3. Benewens die salarisskaal soos aangedui is 'n lewenskostetoelae teen tariewe wat van tyd tot tyd deur die Administrateur vasgestel word, betaalbaar aan voltydse beamptes en werknemers. Die teenswoordige tarief is £320 per jaar vir getroude en £100 per jaar vir ongetroude persone.

4. (*a*) Die Gesamentlike Mediese Personeel word vereis om die Provinsiale Administrasie van die Kaap die Goeie Hoop en die Universiteit van Kaapstad, gesamentlik te dien.

(*b*) 'n Sessie is vier uur per week in verband met kliniese en of opleidingswerk maar is nie noodwendig onafgebroke nie.

5. (*a*) Kandidate moet meld of hulle in aanmerking geneem wil word vir:

(i) aanstelling in 'n voltydse hoedanigheid; of

(ii) aanstelling in 'n deeltydse hoedanigheid.

(*b*) As hulle in aanmerking geneem wil word vir aanstelling in 'n deeltydse hoedanigheid, moet die maksimum aantal sessies wat hulle by aanstelling gewillig sal wees om te onderneem vermeld word.

6. Kandidate moet geregistreerde spesialiste wees in die spesialiteit waarin die vakature bestaan.

7. Die suksesvolle kandidaat indien nie reeds in die Hospitaalraadsdiens nie, moet bevredigende geboorte- en gesondheidsertifikate indien.

8. Aansoek moet gedoen word op die voorgeskrewe vorm (Staf 23), wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, of by die Mediese Superintendent van enige Provinsiale Hospitaal of by die Sekretaris van enige Skoolraad in die Kaapprovinsie.

9. Die ingevulde aansoekvorms moet aan die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, gerig word en moet hom nie later as 22 Januarie 1954 bereik nie. Kandidate moet die vroegste datum meld waarop hulle diens kan aanvaar.

(A562863)

Municipal Board of Mombasa

DEPUTY MEDICAL OFFICER OF HEALTH

Applications are invited for the above post.

Salary £1,200x£50 to £1,450 per annum plus the following allowances:

(*a*) Cost of living allowance: at present £350.

(*b*) Housing allowance.

(*c*) Mileage allowance for use of own car on duty.

Applicants must be registered medical practitioners holding D.P.H. or equivalent qualification. Experience in Infant Welfare and Tuberculosis Control desirable.

Full particulars on request.

Applications in own handwriting, stating date and place of birth, details of qualifications and experience and copies of testimonials should be sent to the undersigned.

A. V. Ratcliff
Town Clerk

P.O. Box 440
Mombasa

LOCUM WANTED

From June 1954 to June 1955, resident doctor required for Mission Hospital for non-Europeans, in the Lowveld, 21 miles from Tzaneen. Hospital 100 beds. X-rays. Nurse-training centre. Surgical and obstetrical experience required. Salary £100 per month. Doctor's house. Apply: Dr. Paillard, P.O. Shiluvane, via Letaba, N. Transvaal.

Provinsiale Administrasie van die Kaap die Goeie Hoop

— HOSPITAALDEPARTEMENT
HOSPITAALRAADSDIENS: VAKATURE

1. Aansoeke word ingewag om die volgende vakante pos:

Inrigting	*Pos*	*Emolumente*	*Sluitings-datum*	*Aansoeke moet gerig word aan*
Conradie-hospitaal, Pinelands	Geneesheer, Graad A	£500–600–660–720 p.j.	8.1.54	Die Mediese Superintendent, Conradie-hospitaal, Pinelands.

2. Die diensvoorwaardes word voorgeskryf ingevolge die Ordonnansie op Hospitaalraadsdiens nr. 19 van 1941, soos gewysig, en die regulasies wat daarkragtens opgestel is.
3. Benewens die salarisskaal soos aangedui is 'n lewenskostetoelae betaalbaar aan voltydse beamptes en werknemers teen bedrae wat van tyd tot tyd deur die Administrateur vasgestel word.
4. Die geslaagde kandidaat, indien nie reeds in die Hospitaalraadsdiens nie, moet bevredigende geboorte- en gesondheidsertifikate indien.
5. Aansoek moet gedoen word op die voorgeskrewe vorm (Staf 23) wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, of by die Mediese Superintendent van enige provinsiale hospitaal of by die Sekretaris van enige Skoolraad in die Kaapprovinsie.
6. Kandidate moet die vroegste datum meld waarop hulle diens kan aanvaar. (A562871)

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT
HOSPITAL BOARD SERVICES: VACANCY

1. Applications are invited for the following vacant post:

Institution	*Post*	*Emoluments*	*Closing Date*	*Applications must be addressed to*
Conradie Hospital, Pinelands	Medical Practitioner, Grade A	£500–600–660–720 p.a.	8.1.54	The Medical Superintendent, Conradie Hospital, Pinelands.

2. The conditions of service are prescribed in terms of Hospital Board Service Ordinance No. 19, of 1941 as amended, and the regulations framed thereunder.
3. In addition to the scale of salary indicated a cost of living allowance at rates prescribed from time to time by the Administrator is payable to whole-time officials and employees.
4. The successful candidate, if not already in the Hospital Board Service, will be required to submit satisfactory birth and health certificates.
5. Application must be made on the prescribed form (Staff 23) which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.
6. Candidates must state the earliest date on which they can assume duty. (A562871)

GROOTHOEK-SENDINGHOSPITAAL

Aansoeke word ingewag van geregistreerde geneeshere vir aanstelling as voltydse inwonende geneesheer. Salaris £1000 plus lewenskostetoelae. Die aanstelling is tydelik vir een jaar waarna dit permanent gemaak mag word. Verdere besonderhede kan verkry word van die Superintendent, Groothoek-Sendingoshpitaal, Zebediela, P/s Naboomspruit, Tvl.

HONORARY OPHTHALMOLOGIST REQUIRED

Applications are invited for the appointment of an Honorary Ophthalmologist to the St. John Eye Hospital. Such applications should be submitted to the Secretary, P.O. Box 7137, Johannesburg.

Transvaalse Provinsiale Administrasie

VAKATURES BY PUBLIEKE HOSPITALE

Aansoeke word ingewag van kandidate met geskikte kwalifikasies vir die onderstaande poste by Publieke Hospitale in die Transvaal.

Aansoeke moet gerig word aan die Geneeskundige Superintendent of Verantwoordelike Geneesheer van die betrokke hospitaal en moet volle besonderhede bevat aangaande die ouderdom, professionele, akademiese en taalkwalifikasies, ondervinding en huwelikstaat van die applikant en moet voorts 'n aanduiding bevat van die vroegste datum waarop diens aanvaar kan word:

Lewenskostetoelae tans betaalbaar aan voltydse werknemers:

	Salaris	*Lewenskostetoelae* *Getroud*	*Ongetroud*
Oor £350	.. .. ..	£320 p.j.	£100 p.j.

Van persone wat aangestel word, sal verwag word om bevredigende sertifikate in te dien, asook om hulle te onderwerp aan 'n geneeskundige ondersoek by die betrokke hospitaal.

Aansoekvorms is verkrygbaar van enige Transvaalse Publieke Hospitaal of die Provinsiale Sekretaris, Afdeling Hospitaaldienste, Posbus 2060, Pretoria.

Benewens jaarlikse salaris en lewenskostetoelae ontvang voltydse werknemers spoorwegkonsessie en word verlof toegestaan ooreenkomstig die hospitaal verlofregulasies.

Die sluitingsdatum van aansoeke vir die poste is 11 Januarie 1954.

Hospitaal	*Pos*	*Salaris*	*Opmerkings*
Johannesburg Hospitaalbestuur en die Universiteit van die Witwatersrand	Ortopediese Chirurg	£1,800	Hoër kwalifikasies in Chirurgie 'n vereiste.
Klerksdorp	Radioloog	£1,800	Gekwalifiseerde Radioloog. Moet diens doen te Klerksdorp) Potchefstroom en Wolmaransstad Hospitale.
Vereeniging	Neuro Psigiater	£205	Geregistreerde Mediese Praktisyn. Moet behoorlik deur opleiding en ondervinding gekwalifiseerd wees.

43840

Printed by National Commercial Printers, Elsiesriver, and Published by the Proprietors, *The Medical Association of South Africa, Medical House*, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'